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THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES.

DECEMBER, 1905.

XANTHELASMA AND CHRONIC JAUNDICE.[1]

By Thomas B. Futcher, M.B. (Tor.),
ASSOCIATE PROFESSOR OF MEDICINE, JOHNS HOPKINS UNIVERSITY, BALTIMORE, MD.

(From Prof. Osler's Clinic, Johns Hopkins Hospital.)

Xanthelasma, xanthoma multiplex, or vitiligoidea as an accompaniment of chronic jaundice is a comparatively rare affection. This paper is based on three such cases admitted to Dr. Osler's wards at the Johns Hopkins Hospital. These constitute all the cases that have occurred in the hospital during the first sixteen years ending with May 15, 1905. During this period there had been 18,400 medical admissions, among which there was a large number with chronic icterus. All three cases were admitted between January 30 and June 26, 1901, two having been under observation in the same ward at the same time. The main points in the clinical histories of the cases follow:

Case I. (*Gen. Hosp. No.* 33,689).—*History of typhoid fever; later, cholecystitis; onset of symptoms of gallstones of the common duct two years after the typhoid infection; eight months after the onset of the jaundice the xanthoma plaques made their appearance; xanthomata on gums; exploratory laparotomy; gallstones in the gall-bladder and common duct; spontaneous disappearance of the xanthelasma after leaving the hospital.*

L. J. F., female, white, married, aged thirty-nine years, was admitted to the Johns Hopkins Hospital on January 30, 1901, complaining of "jaundice."

The family history was unimportant, with the exception that her father had had three attacks of jaundice associated with enlarged liver, attributed to the use of alcohol.

[1] Read before the Association of American Physicians, Washington, D. C., May, 1905.

The patient was healthy as a young woman. Married twenty-one years. One child eighteen year ago. Typhoid fever in June, 1897, about three and one-half years before admission. Attack lasted five weeks. After temperature returned to normal, but before she was out of bed, she had a very severe attack of pain in the epigastrium, associated with vomiting. There was great sensitiveness in the upper abdomen, with fever of several days' duration.

The present illness dated from the summer of 1899, two years after the typhoid infection. It began with an attack of abdominal pain, without jaundice. In December, 1899, thirteen months ago, she had several attacks of colicky pain in the epigastrium, after which she became jaundiced. These were accompanied by chills, fever, and sweating. The epigastrium was described as being swollen and very sensitive. From that date there had been repeated attacks of the same character. Jaundice had been persistent, but varying somewhat in intensity. The stools were intermittently clay-colored. Patient lost fifty-three pounds in thirteen months.

The jaundice had existed for thirteen months. There had been severe pruritus. Five or six months ago, and eight months after the onset of the jaundice, she first noticed flat, yellow patches appearing on the palms of the hands, particularly in the folds of skin at the flexures of the joints. Later others appeared in the folds of the elbows, over the olecranon processes, axillæ, sides of the neck, and dorsal surfaces of the toes. At first flat, they subsequently became somewhat raised. The patches over the olecranon processes were sensitive on being struck, and those on the hands were also tender when she used the broom. Friction seemed to favor their occurrence. One particularly thick xanthoma patch developed beneath a ring on the third finger of the left hand. The finger became so sensitive that the ring had to be removed.

Physical Examination. The following were the essential features in the physical examination on admission: Patient still fairly well nourished. Uniformly jaundiced; sclerotics deep yellow in color.

Distribution of Xanthoma Plaques. One very small one on left upper eyelid. Large, flat, yellow patch on right side of neck, 3 x 4 cm. in diameter, with small outlying portions. Similar but still larger one on right side of neck. Smaller one over the fold of left trapezius muscle. *Right arm and hand.* Patch in fold of axilla. Areas of keratosis about elbow and numerous nodular xanthomata around them. Exactly corresponding to the fold of the elbow there was a long linear xanthoma, with small outlying ones. Small patch over one of the creases at the wrist. Two in the palm of the hand over palmar creases. In the creases on the flexor surfaces of all the metacarpo-phalangeal and nearly all the interphalangeal joints there were xanthoma plaques, usually linear, but occasionally irregular in outline. A few patches were scattered over the sides of the fingers and on the knuckles. *Left arm and hand.* The distribution of the

lesions was so nearly identical with those on the right upper extremity that a separate detailed description is unnecessary. A few patches on the upper chest. None on abdomen. Few on dorsal surface of the toes. All the patches had a distinct whitish-yellow tint, differing materially from the color of the jaundiced skin. The majority of the xanthomata were flat and not elevated. A few of the older ones were elevated and slightly nodular, particularly those on the extensor surfaces of the elbows.

The examination of the thoracic organs revealed nothing of importance. The liver was considerably enlarged. The absolute hepatic flatness extended 6.5 cm. below the costal margin in the mammillary line, and 13.5 cm. below the tip of the ensiform in the median line. The gall-bladder was not palpable. The splenic border was rounded and palpable 5.5 cm. from the costal margin.

The stools were light in color, but contained some bile. Urine contained bile pigments. Blood coagulation time on admission was eight minutes and fifteen seconds.

On January 31st Dr. Osler made the following notes: "Interesting points in connection with the case are: (1) sequence of typhoid, cholecystitis, and gallstones; (2) chronic jaundice associated with delayed coagulation time; (3) chronic jaundice associated with the symptom-complex of gallstone in the common duct; (4) xanthelasma.

On February 1st I noted that there was apparently the same sort of lesion on the gums as was found on the extremities. The patches had an opaque yellow appearance and were slightly thickened. The lesion was most evident on the gum over the upper incisors and on that over the lower incisors and canines.

On February 27th a few fresh xanthoma patches were noted for the first time over the patient's back. Under the administration of calcium chloride the coagulation time of the blood became reduced to three minutes and fifty-five seconds.

Operation. On February 28th Dr. Halsted performed an exploratory laparotomy, and found two large stones in the gall-bladder and one in the common bile-duct. There were no indications of xanthomata in the mucous membrane of the gall-bladder or bile-duct.

The patient did well until the eighth day, when she had a severe secondary hemorrhage from the wound. Later she developed a fibrinous pleurisy and a pericarditis, from which she eventually recovered, and was discharged from the hospital much improved, but still somewhat jaundiced, on June 1, 1901.

Dr. Florence Sabin, who was Medical Interne in charge of the patient when she was in the hospital, followed the subsequent progress of the case and she has kindly turned over the notes to me. These may be briefly summarized as follows:

April 7, 1903. General health excellent; still jaundiced. Operation wound healed after eighteen months. Had occasional attacks

of soreness over the liver region, which lasted a few days, during which time the urine becomes very dark.

March 9, 1905. Patient wrote that she was in excellent health and weighed 150 pounds. In reply to a question as to the present condition of the yellow patches she stated that they had entirely disappeared.

In this case the xanthomata were mainly of the flat variety, although there were a few of the nodular variety about the right olecranon. The jaundice had been present for eight months before the onset of the xanthomata. An interesting feature of the case was the spontaneous disappearance of the xanthomata after the patient left the hospital.

CASE II. (*Gen. Hosp. No.* 33,912).—*Gallstones of the common and hepatic ducts and of the gall-bladder; biliary hypertrophic cirrhosis; jaundice of ten years' standing; appearance of xanthomata eight years after the onset of the jaundice. Severe hemorrhages from the nose and uterus; death and autopsy.*

M. A. C., female, white, married, aged thirty-nine years, was admitted to the Johns Hopkins Hospital on February 19, 1901, complaining of hemorrhages.

The family history was unimportant. The patient had always been a healthy woman previous to the onset of the present illness. She was the mother of six children.

The present illness began ten years ago with what she described as "malaria." She had attacks characterized by chills, fever, sweating, and paroxysmal pain in the right hypochondrium at the beginning of the attacks. Jaundice developed at the onset and has since continued with varying degrees of intensity. The attacks of pain, chills, and fever have recurred at intervals up to the present. In March, 1900, she had severe hemorrhages from the nose and uterus, which lasted three weeks. Four days before entrance these recurred and became so severe that she sought admission to the hospital for them. For years "blood blisters" the size of a split pea had appeared over the body, and she was liable to have bruises develop on the slightest injury.

Two years ago, and eight years after the onset of the jaundice, she noticed yellow patches developing on the palms of the hands and subsequently on other parts of the body. There were no symptoms referable to them.

Physical Examination. The following were the essential features in the examination: The patient was fairly nourished. There was intense jaundice and extreme pallor of the skin and mucous membranes. The patient was very freckled. The lungs were clear on percussion and auscultation. The heart showed nothing of importance excepting a faint systolic murmur, probably anæmic in origin, at the mitral area. The relative liver dulness began at the fifth rib in the mammillary line; the absolute at the sixth rib in the same line

not extending quite to the costal margin. Just below the costal margin and inside the mammillary line there was an indefinite rounded mass to be felt, which descended on inspiration and suggested possibly a distended gall-bladder.

Xanthomata. On both of the upper and lower eyelids were long, somewhat raised yellow plaques. In the lines of the palmar surface of both hands and along the sides of the fingers were similar plaques, only less raised. The elbows, both flexor and extensor surfaces, and the axillæ had small rounded tumor-like nodules. Over both buttocks were numerous larger, round, raised, nodular, yellow masses, varying up to 0.5 cm. in diameter. There were a few over the knees and dorsal surfaces of the feet.

There was a spontaneous subcutaneous hemorrhage over the right knee, and a continuous flow of pale bloody fluid from the vagina.

The urine contained abundance of bile pigments, a trace of albumin, but no casts.

On admission the red cells were 1,880,000 per c.mm.; the leukocytes, 15,700 per c.mm.; and the hæmoglobin, 23 per cent. The coagulation time was fourteen minutes. Under complete rest and the administration of iron the patient's condition markedly improved. On March 25th the blood count was as follows: red cells, 4,804,000 per c.mm.; leukocytes, 9700 per c.mm.; hæmoglobin, 58 per cent. Under calcium chloride the coagulation time on this date had become reduced to four minutes and fifty-three seconds. Her condition continued to improve and she progressively gained in weight. She refused an exploratory operation, and, at her own request, was discharged on April 18, 1901.

A few days after her discharge from the hospital the patient died, death having been preceded by severe uterine hemorrhages. Permission was obtained for an autopsy, which was made by Dr. W. G. MacCallum.

Anatomical Diagnosis. Cholelithiasis; obstructive jaundice; biliary hypertrophic cirrhosis of the liver; splenic tumor; acute nephritis; ovarian cystomata; xanthelasma multiplex.

The following were the chief features about the liver and bile passages: The common duct was plugged with a large plum-shaped gallstone. There were several gallstones in the gall-bladder, almost embedded in saccules in the bladder wall. The hepatic duct was filled by a curved cylindrical concretion. Above this the gall-ducts throughout the liver were enormously thickened. The walls of the ducts were almost 0.5 cm. in thickness and of a deep yellow color. On section of the liver the bile-ducts stood out everywhere looking like sclerotic arteries. There were some minute yellow flecks in the mucosa of the walls of the bile-ducts, but there were no lesions typical of xanthomata. The liver was large and measured 35 x 18 x 11 cm., and weighed 2800 grams.

The chief features of interest in this case were: 1. The combination of cholelithiasis, with biliary hypertrophic cirrhosis as a cause of the jaundice. 2. The development of the xanthomata only after the jaundice had existed for eight years. 3. The xanthomata were of two types—flat and nodular.

CASE III. (*Gen. Hosp. No.* 35,296).—*Hypertrophic cirrhosis of the liver; severe uterine hemorrhages; jaundice lasting one and a half years before the onset of the xanthomata; hemorrhages from the gums and uterus.*

L. H., female, white, married, aged forty-two years, was transferred from the surgical to the medical department of the Johns Hopkins Hospital on June 26, 1901, complaining of "liver trouble and jaundice."

With the exception of her father and mother having both died of tuberculosis the family history was unimportant. The patient had had six children and one miscarriage. She had been in the habit of taking an occasional glass of beer or porter for years; no whiskey. For several years she had suffered from attacks of abdominal pain, particularly after indiscretions in eating.

She dated her present illness from five years ago, when she began to have vertigo and general weakness and was forced to give up her work. Her bowels, which had been previously constipated, became rather loose. Two and one-half years ago jaundice developed, which had continued ever since. The stools were gray and the urine high colored. There had been no chills, fever, or attacks of abdominal colic, although she had had indefinite pains in the left hypochondrium. For a year she had noticed a tender spot over the liver, the sensitiveness increasing with pressure or deep inspiration. For the same period she had suffered from repeated attacks of severe uterine hemorrhages. On November 19, 1900, she was admitted to the surgical side of the hospital. She was then jaundiced. The red blood cells were 1,504,000, per c.mm., and the hæmoglobin 28 per cent. The coagulation time was between three and four minutes. Owing to the severe anæmia an exploratory laparotomy seemed inadvisable. Her general blood condition gradually improved, and on May 31, 1901, Dr. Mitchell performed an exploratory laparotomy. Careful examination of the bile passages failed to reveal any gallstones. The liver was much enlarged and presented the appearances of an hypertrophic cirrhosis, which was the surgical diagnosis. The post-operative history was uneventful, and the wound healed well. The jaundice persisted, however, and on June 26, 1901, she was transferred to the medical side of the hospital.

For about one year previous to her admission to the medical ward she had noticed whitish-yellow spots appearing, one below the left eye, the other below the right. These had been gradually increasing in size.

Physical Examination. The patient was of small stature, but was fairly well nourished. Jaundice was intense. The lungs were

normal. Over the apex of the heart was heard a loud blowing systolic murmur, which was transmitted to the axilla, and was also audible over the body of the heart. The liver dulness was considerably increased and extended about 6 cm. below the costal margin in the right mammillary line. The border could not be distinctly felt, owing to the tenderness over the hepatic region. There was considerable tympanic distention of the abdomen; but no ascites. The spleen was not palpable.

Situation of the Xanthomata. Over the lower right eyelid there was a flat yellow xanthoma patch measuring 1 cm. x 2 mm. Above and within the left internal canthus there was a similar one measuring 1.5 cm. in diameter. Careful search failed to reveal any xanthomata on any part of the body.

The urine contained abundance of bile pigments, but no albumin or casts were present.

During her stay on the medical side she had some bleeding from the lips and mucous membrane of the mouth. On July 4, 1901, she was discharged from the hospital at her own request.

The patient returned to the hospital for observation on November 28, 1904. The jaundice was still present. She had had some bleeding from the gums and from a scratch on the ear. The liver border was palpable three fingers' breadth from the costal margin.

In this case the xanthomata were confined to the eyelids. The patient gave a definite account of their appearing one and a half years after the onset of the jaundice. The case was therefore not one in which a patient with xanthomata of the lids developed a chronic jaundice, but the reverse. Although the xanthomata were not so widely distributed as in the previous cases, yet there was no reason to doubt that they were dependent on the hepatic disease or the jaundice.

Xanthelasma is not a common disease under any circumstances. The cases in which it is limited to the eyelids (*xanthoma palpebrarum*) occur much more frequently than those where the lesions are more generally distributed (*xanthoma multiplex*). The majority of the latter form are of congenital origin in the young, or associated with jaundice or glycosuria in adults.

Xanthomata occur in two forms, either as flat plates or plaques (*xanthoma planum*), or as nodules or tumors (*xanthoma tuberculatum* or *tuberosum*). According to Crocker they represent little more than differences in position, shape, and degree of development.

Xanthoma palpebrarum constitutes the bulk of the cases, and the lesion here is in the form of *plaques*. It commences most frequently on the left upper eyelid near the inner canthus, and may form a semicircle of the whole eye. Later the right side becomes affected and the lesion is usually symmetrical. The patches have a chamois-leather yellow color. They are either not at all or only slightly raised

and are situated in the corium. They are soft and smooth and the skin does not seem thickened when it is picked up.

Xanthomata when of the *nodular* variety have the same color as the plaques, but project more or less above the surface. They vary in size from a millet-seed to a large pea, and in rare instances may be 2 or 3 cm. in diameter. The small ones are convex, round or oval, and often have a fine network of capillaries over them. They are soft and smooth to the touch. The larger ones are made of conglomerations of the smaller ones, and are irregular in contour and are more or less firm in consistence, depending upon the amount of connective tissue they contain.

Xanthoma multiplex in the adult is most frequently associated with jaundice of long standing, and it is this form with which we are especially interested in this paper. The first reference I have been able to find of the association of xanthelasma with chronic jaundice is in a paper by Thomas Addison and William Gull,[1] published in 1851, and entitled "On a Certain Affection of the Skin, Vitiligoidea—α, Plana, β, Tuberosa. With Remarks and Plates." They report five cases of a skin affection with which they were not familiar, and, owing to a certain resemblance between it and the disease described by Willan as vitiligo, they suggested the name *vitiligoidea*. They distinguished two varieties of the affection: the flat, to which they gave the name *vitiligoidea plana*, and the nodular, which they designated by the name *vitiligoidea tuberosa*. Of the five cases three were in patients with chronic jaundice. In one of the three the lesions were distributed in the creases of the wrists, hands, and interphalangeal joints, similar to the distribution in Cases I. and II.

The lesions of xanthoma multiplex in chronic jaundice are both in plaques and nodules, the former type prevailing. They may involve the skin, mucous and serous membranes, and the tendons. The commonest positions are the eyelids, where it is said to most often begin, the palms and soles and backs of the hands and feet, flexures of the wrists, and the buttocks in and near the cleft and about the anus. It is of interest to note that in Cases I. and II. the xanthomata made their appearance first on the palms of the hands. The plaques are most commonly found on the eyelids, joint flexures, and mucous membranes. In Case I. the gums were affected. In Case II. there were suggestive indications of xanthomata in the mucous membrane of the bile-ducts on macroscopic examination, a suspicion which was confirmed on microscopic study of the sections. The nodules usually occur on the extensor aspects of the joints, particularly on parts exposed to irritation, as the knuckles, elbows, and knees. In xanthoma multiplex symmetrical arrangement of the lesions is the rule, and is often very striking. This was a marked

[1] Guy's Hospital Reports, new series, 1851, vol. vii. p. 265

feature of Cases I. and II., and was also true in Case III., in which the lesions were limited to the eyelids. The xanthomata appear gradually and are slow in their development. After months or years their growth ceases, and there is usually no future change in them. Crocker states that three instances have been reported in which spontaneous disappearance occurred after several years. In Case I. the xanthomata had entirely disappeared within four years after the patient left the hospital, and about four and a half years after their first appearance.

The xanthomata rarely cause any inconvenience excepting from their disfigurement. Occasionally, burning, prickling, or itching have been experienced, and when they have occurred on the palms of the hands or knees, grasping objects or kneeling may cause some discomfort, or even pain.

In 1882 a committee composed of Jonathan Hutchinson, Alfred Sangster, and H. Radcliffe Crocker[1] was appointed by the London Pathological Society to report on 2 cases of xanthoma multiplex, one of which was demonstrated before the Society by Stephen Mackenzie, and the other by James Startin. They collected data of 23 cases of xanthoma multiplex in adults with jaundice; 10 of these were in males, 12 in females, and in 1 the sex was not stated. It would seem, therefore, that women are a trifle more liable to the affection than men. They also found records of 5 cases of xanthoma multiplex in adults without jaundice; 4 of these were males and 1 a female. Statistics of 8 cases of xanthoma multiplex, either congenital or commencing before puberty, were also collected from the literature. It was not possible to definitely ascertain the sex in these cases, but 4 were believed to be males and 4 females.

Some of the conclusions of this committee regarding xanthoma multiplex were as follows: 1. That cases occurring before puberty, although structurally the same as those beginning at a later period, are etiologically different, have no traceable connection with hepatic disease, but are probably to some extent hereditary. That they may be congenital, and that the eyelids always escape. 2. That four-fifths of the cases of xanthoma commencing after puberty are associated with chronic jaundice from obstruction of the bile-ducts. That in some of the others the health has been perfect, but that diabetes has been observed more than once. That the eyelids are almost always involved and almost invariably the starting point. Occasionally spontaneous involution occurs. That, as regards sexes, the numbers affected are nearly equal.

From the standpoint of *etiology* there seems little doubt but that the jaundice is the exciting agent in the xanthoma multiplex cases accompanied by this symptom. The hepatic conditions causing the

[1] Transactions of the Pathological Society of London, 1882, vol. xxxiii. p. 376.

jaundice have been found to be cholelithiasis, stricture of the bile-ducts, atrophic and hypertrophic cirrhosis of the liver, hydatids, and cancer. How the jaundice acts in effecting the changes in the corium is not known, but presumably it is through the agent of a toxin. Besnier, Hollopeau, and Kaposi regard the jaundice and liver troubles as secondary, and due to the development of the xanthomata in the viscera. Crocker states that the order of development of the clinical features does not support this view. It may be of interest here to note that Besnier and Gailleton have described a condition in hepatic affections which they call *xanthochromia* of the skin, and not due to jaundice in some cases. It is more marked on the face and trunk than on the extremities, the conjunctiva and buccal mucous membrane are uncolored, and there is no bile in the urine and feces.

There is considerable difference of opinion as to the *pathology* of this skin affection. Xanthomata were excised from Cases I. and II., and the sections were carefully studied by Dr. W. W. Williams, of the Harvard Medical School, whose report has not yet appeared, but who has kindly furnished me with a copy of his histological examination. In association with these cases, sections from a xanthoma palpebrarum complicating a case of diabetes insipidus, admitted to the Johns Hopkins Hospital and reported by me before this Association last year, were also studied. The findings in all three cases were practically identical, and I quote briefly the main features of Dr. Williams' report:

"The epidermis is essentially normal, although in places there are large vacuolated cells in the lower part. Below this is a layer of practically normal connective tissue, which, however, contains a few pigmented spindle-shaped cells. Branching off from this layer are numerous larger and smaller fibrous bands, becoming continuous with the normal tissue deeper down, and forming an irregular network. These trabeculæ are composed of fibrous connective tissue, with a few deeply staining, long, narrow nuclei and some delicate elastic fibres. Also a few delicate pigment granules are to be seen.

"Within the meshes of this fibrous reticulum are masses of pale cells, although in places the same pale cells occur singly or in pairs between the connective-tissue fibres. Surrounding each cell in these groups is a delicate reticulum containing here and there fine elastic fibres.

"These pale cells (so-called xanthoma cells) vary much in size; and though some are small and mononuclear and others large and multinuclear, they are all otherwise similar. There is no definite cell membrane, and the protoplasm is everywhere finely granular and contains more or less refractive granules, which react to the fat stains. Some of the cells are vacuolated, and others definitely phagocytic.

"The nuclei are, as a rule, oval, with a distinct membrane, vesicular, and often contain several nucleoli. They are usually more or less centrally situated. Mitosis is seen in some of these pale granular cells. The number of nuclei vary from one to twenty.

"About some of the bloodvessels, traversing these groups of cells, slight lymphoid cell infiltration has taken place. The endothelium of some of these vessels and of many lymph spaces is swollen and contains fatty granules. Also in the perivascular lymph spaces are seen pale, granular, vesicularly nucleated cells. In none of the specimens were any polymorphonuclear leukocytes, nerves, or muscle cells found, or any involvement of the hair follicles or sebaceous or sweat glands noticed."

Dr. Williams' report on the histological examination of the bile-ducts in Case II., which came to autopsy, is exceedingly interesting. "The walls of the bile-ducts are considerably hypertrophied, containing many elastic fibres, and the lumena are lined with a mass of lymphoid and plasma cells, and also many large, pale, granular, vesicularly nucleated cells, similar to those already described in the skin lesions." Apparently these changes were regarded as due to the development of xanthomata in the bile-ducts.

In a communication from Dr. Williams he wrote: "In my conclusions on these growths I maintained that they were of endothelial origin, though many described in the literature, and also one I described as really lipomata—lipoma xanthomatodes."

The description given by Crocker[1] and Touton for sections of xanthoma palpebrarum agree closely with Williams' report. There is no essential difference between the plaques and the nodular variety. In the latter the bulk of the lesion is more superficial, and is in the papillary layer pushing up the epidermis above the level of the surrounding surfaces. The connective tissue is much more abundant than in the case of the plaques, and is most marked about the hair follicles and sebaceous glands. The fatty masses are less conspicuous and yellow oil globules infiltrate the meshes between the fibrous tissue. Crocker holds that in the development of the xanthomata inflammation is the primary factor; that the xanthoma cells and the connective-tissue growth are secondary, and that the whole process is of toxæmic origin.

Köbner thinks that the lesions are derived from embryonic remnants. Török claims that the xanthoma plaque is composed of adipose cells interrupted in their progress to complete evolution, and that it is not a tumor, but an excess of growth. Unna holds that the fat in xanthoma palpebrarum is a sort of fatty infiltration of the orbicularis muscle, the fat being in the lymph spaces and the giant cells being sections of dilated lymphatics. Pollitzer claims that the eyelid lesion is the product of the degeneration of embry-

[1] Diseases of the Skin, third edition, p. 745.

onically displaced muscle fibres. This view has not been supported and seems to be disproven by the clinical fact that in both children and adults, after the lesion begins on the eyelids, it subsequently makes its appearance on various parts of the body.

There is no special difficulty in making a *diagnosis*. About the only condition likely to be confused with it is *milium palpebrarum*. These are usually small and situated near the inner canthus of the upper lid. Being due to retained sebaceous secretion, they are generally white, and occasionally the dark-colored opening of the duct may be seen. Milium is more superficial and firmer, and the contents of the nodule can easily be peeled out. Politzer refers to cases of *multiple dermoid cysts* of the skin, in which the diagnosis was possible only on microscopic examination of excised tissue.

After developing gradually for years the xanthomata become stationary and persist in the majority of cases throughout life. In rare instances, as in Case I., spontaneous involution may occur. In this case operation had been performed with a relief of the gallstone symptoms and almost complete disappearance of the jaundice.

There is practically no treatment for the condition, excepting incision, that yields results. It seems almost certain that the xanthomata are due to the jaundice or to some associated toxic substance circulating in the blood. Consequently efforts should be made to relieve the causes of the jaundice, which is possible at least in the cases of cholelithiasis.

SUMMARY. The three cases here reported were all in women, their respective ages being thirty-nine, thirty-nine, and forty-two years. The duration of the jaundice before the xanthomata appeared was eight months, eight years, and one and one-half years. The cause of the jaundice was gallstones of the common duct in two cases, one of which also had biliary hypertrophic cirrhosis of the liver, and in the third case hypertrophic cirrhosis of the liver alone. In all three cases the eyelids were involved, in one these only. In the other two there were lesions on the extremities and to a less degree on the trunk. In two cases the xanthomata appeared on the hands first; in the third on the eyelids. The xanthomata were chiefly in the form of plaques in all the cases. There were a few nodular xanthomata in Cases I. and II. The distribution was strikingly symmetrical. In Case I. there were xanthomata on the gums, and in Case II. histological examination of the bile-ducts showed that xanthomata of the mucous membranes of the ducts were present. At all events, the histological picture showed cells identical with the "xanthoma cells" of the lesions in the skin. In Case I. there was a spontaneous disappearance of the xanthomata four and one-half years after their onset, and about four years after the gallstones were removed at operation. Two of the cases were operated on, and one came to autopsy, so that the causes of the jaundice were definitely ascertained.

Four-fifths of the cases of xanthoma multiplex in adults occur in chronic jaundice. In these cases men and women are about equally affected, with possibly a slightly greater frequency in the latter. The hepatic conditions causing the jaundice have been found to be gallstones, stricture of the bile-ducts, atrophic and hypertrophic cirrhosis of the liver, hydatids, and cancer. In addition to the skin, the tendons and mucous membranes may occasionally be involved. The onset is said to be most frequently on the eyelids. The xanthomata accompanying jaundice are believed to be directly traceable to the latter or to some associated toxic substance circulating in the blood. Symmetry in distribution is often striking, and the tendency to develop in the folds and creases of the joints is a remarkable feature. The views as to the actual nature and sequence of tissue changes in the lesions vary much. Crocker thinks that inflammation is the primary factor and that the xanthoma cells and connective-tissue growth are secondary, and that the whole process is of toxæmic origin. Williams' careful histological study indicates strongly that the xanthomata are of endothelial origin. Once they develop they rarely disappear. Spontaneous involution is rare. No treatment other than surgical in suitable cases is of any avail. The indications are to relieve the underlying process if possible.

THE NATURE AND THE LESIONS OF CIRRHOSIS OF THE LIVER, WITH SPECIAL REFERENCE TO THE REGENERATION AND REARRANGEMENT OF THE LIVER PARENCHYMA.[1]

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The study of cirrhosis of the liver has been approached from several points of view—etiological, anatomical, and clinical; but since the disorder was first described by Vesalius special interest has centred in the obvious and most conspicuous feature of the lesions—the fibrosis; so much so, indeed, that the term "cirrhosis," first employed by Laennec to describe the yellowish (*κιρρός*) "hob-nails," which he regarded as new-growth, has by a curious transference of meaning become almost inseparable from, with some writers practically identical with, the sclerotic process; and, indeed,

[1] Read before the Association of American Physicians, Washington, D. C., May, 1905.

by the ill-informed its meaning is not infrequently expanded so as to include sclerotic and fibrotic processes in other organs. As contrasted with the conspicuous fibrosis, the changes in the parenchyma of the liver in cirrhosis have received comparatively scant attention; they are commonly, though of course not universally, looked upon as progressively and continuously degenerative or atrophic, usually subsidiary in importance, and secondary to, consequent upon, the changes in the interstitial tissue. It is true, however, that the so-called pseudobile-ducts or canaliculi, present in many cases of cirrhosis, are usually nowadays looked upon as evidences of a regenerative process; that the liver cells are further capable of quite extraordinary regeneration is now well known in consequence of experimental investigations and the study of the changes following acute yellow atrophy and allied processes, and such regeneration is generally recognized in the adenoma-like formations that occur under diverse circumstances; but that any noteworthy regeneration of the liver cells is a part of ordinary cirrhosis is not commonly taught.

It has long been recognized, however, that much as we have felt that we know concerning the liver and its perversions of function and alterations of structure, much more remains to be learned; though the clinical recognition of cirrhosis of the liver may be a comparatively easy matter in many cases, the condition is not infrequently overlooked, and it constitutes a not uncommon accidental finding at the necropsy; and the divers classifications of the process (or processes) proposed by different writers—some based largely, others exclusively, upon the nature, disposition, arrangement, etc., of the fibrous tissue—and the discussion relative to what is and what is not cirrhosis (in an acceptable sense) indicate only too well that many questions pertaining to the subject still await solution. The general obscurity of many of these questions was well emphasized by Dr. Cheadle[1] in his Lumleian lectures for 1900, when he suggested the following as "among the questions awaiting an accepted and final settlement: Is atrophic cirrhosis—that is, the contracted hobnail liver—always of alcoholic origin? Is the liver small from the first, or is there an earlier stage in which it is enlarged, becoming contracted later; or is atrophic cirrhosis merely a late stage of hypertrophic cirrhosis; or is there an alcoholic cirrhosis in which the liver is hypertrophied and enlarged from first to last; or is hypertrophic cirrhosis limited to the biliary cirrhosis of Hanot and Charcot of uncertain origin; or, it is even asked, Does the biliary form described by French authorities exist at all as a distinct entity? Again, is ascites limited to the atrophic form, almost unknown in the hypertrophic; while, on the other hand, is jaundice rare in atrophic cirrhosis, almost invariable in the hypertrophic

[1] Some Cirrhoses of the Liver, 1900.

form? Are the microscopic characters of monolobular cirrhosis, with hyperplasia of the bile-ducts, as described in the biliary cirrhosis of Hanot and Charcot, found only in cases that correspond to that clinical type; or are they met with in both atrophic and hypertrophic cirrhosis of alcoholic origin? The part played by alcohol, syphilis, and malaria in causation and their connection with special types of the affection; the incidence and significance of perihepatitis; the curability of ascites; the efficacy of paracentesis or its uselessness in the treatment of it; the possibility not of curing cirrhosis, but of prolonging the life of the cirrhotic patient by this or other means; the conditions under which the course of cirrhosis can be favorably influenced in this way, are other points about which there is a lack of general agreement."

Although progress has been made since the time of Dr. Cheadle's lectures, which was about contemporaneous with a renewed interest in cirrhosis of the liver, much still awaits elucidation. The difficulty in the past has been due largely to the points of view from which the phenomena of cirrhosis have been studied, to the fact that the late lesions are those that necessarily have been most investigated, to the bases of classifications commonly adopted, and to the comparative inelasticity of the descriptive terms employed, all of which are excellently intimated by Dr. Cheadle in his queries. The recent studies and several communications of Kretz,[1] however, throw considerable light upon the nature of cirrhosis; and the still more recent communications by MacCallum[2] and Böstrom,[3] as well as the results of some experimental work, notably recently by Pearce,[4] lend confirmation to his views. My own work, which in some particulars is merely confirmatory of the already published work of Kretz and of MacCallum, is based upon a study of the thirty cases of cirrhosis and of many other cases of disease of the liver, that have come to necropsy at the German Hospital, Philadelphia, during the last eight years; certain clinical data of patients treated in the German Hospital during the last fifteen years, in whom a diagnosis of cirrhosis of the liver was made—for permission to use which I am much indebted to Dr. James C. Wilson, physician-in-chief to the German Hospital; several cases of cirrhosis observed on Dr. Musser's service in the University Hospital, for permission to utilize which I am much indebted to Dr. Musser; several cases observed on my own service at St. Agnes' Hospital; many of Professor Kretz's

[1] Ueber Lebercirrhose, Wien. klin. Woch., 1900, Bd. xiii. p. 271; Lubarsch und Ostertag's Ergebnisse der allg. Path. und path. Anat., 1901, Bd. viii. p. 473 (bericht über das Jahr., 1902); Verhandl. der Deut. path. Gesellschaft, 1901, Bd. viii. p. 54; International Clinics, 1905, 15th series, vol. iii. p. 289.

[2] Regenerative Changes in Cirrhosis of the Liver, Journal of the American Medical Association, 1904, vol. xliii. p. 649.

[3] Lebercirrhose, Deut. med. Woch., 1905, Bd. xxxi. p. 167.

[4] A paper on Experimental Cirrhosis of the Liver, read at the meeting of the American Association of Pathologists and Bacteriologists, in Chicago, April, 1905, and to be published in the Journal of Experimental Medicine.

preparations; and other pieces of tissue from unusual cases, for which I am indebted to several friends, notably Dr. W. F. Hamilton, of Montreal; Dr. Allen J. Smith, of Philadelphia; Dr. W. M. L. Coplin, of Philadelphia (spontaneous cirrhosis in dogs), etc. I shall consider (1) the structure of the liver in so far as it has a bearing upon the changes in cirrhosis; (2) the changes in the parenchyma of the liver in cirrhosis; (3) the changes in the connective tissue in cirrhosis; and (4) the permissible deductions with regard to the nature of cirrhosis that may be drawn from the foregoing.

THE STRUCTURE OF THE NORMAL LIVER. Although it is largely supererogatory at this day to discuss the structure of the normal liver, a clear conception of the cirrhotic process is best attained by bearing in mind that, as recognized years ago by Hyrtl[1] and recently especially emphasized by Kretz, the liver is not made up of acini in an acceptable sense. While the physiological unit of the liver is that collection of cells drained by a radicle of the bile-duct, this unit is scarcely delimitable anatomically, although Sabourin[2] has made an attempt to do so; nor are the so-called acini, or lobules, cross-sections of which, surrounded by connective tissues, form such a conspicuous feature of microscopic sections, of such simple structure as at first sight appears. Study of the liver in serial sections and its reconstruction show that the liver parenchyma does not form isolated acini, or lobules, everywhere surrounded by connective tissue, but that what appear to be acini are merely cross-sections of liver cells arranged as a mantle about the numerous dendritic ramifications of the hepatic vein, and that this mantle is nowhere discontinuous, forming, on the contrary, a continuous whole, everywhere connected, particularly at the points of junction of the sublobular, lobular, and lobar veins. The intimate relationship that the liver cells structurally bear to the hepatic vein is further shown by the radial disposition of the cells about the branches of this vein—the so-called central veins. Toward the periphery of the liver anastomoses may sometimes be found between branches of the hepatic vein, and in some of the lobules one sometimes encounters rather large branches of the central vein, about which the liver cells are arranged radially. Thus we find that the liver consists not of acini, but of mantles of cells arranged radially about the arborescent ramifications of the hepatic vein, and that cross-sections of so-called acini are really cross-sections of cell mantles, which, however, may also be cut longitudinally or tangentially.

Sabourin has attempted to modify the current conception of the so-called acini in that he looks upon the centre of the acinus, or lobule (according to his conception), as the portobiliary nodule, the point of breaking up of a radicle of the portal vein, and of the

[1] Lehrbuch der Anatomie des Menschen, 1873, 12te Aufl., p. 639

[2] Etude comparé du foie de l'homme et du foie du cochon, Revue de méd., 1901, tome xxi. p 384

confluence of the biliary capillaries to form a biliary radicle; the extent of the acinus, as the area of distribution of a portal radicle, with its accompanying hepatic artery, bile-duct, and related liver cells; and the periphery of the acinus, as the so-called rectilinear veins, small intralobular veins, formed by fusion of capillaries, that empty at right angles into the central veins. In the swine these rectilinear veins are accompanied by connective tissue from the periportal connective tissue to the central veins, and Sabourin believes that the counterpart of this exists in the human subject; it is to be found especially in cases of long standing chronic congestion. This conception of the architecture of the liver is of some interest, since it serves to correlate the anatomical with the physiological unit; but as far as we are concerned at present, it merely substitutes the portal for the hepatic vein, since in both cases the hepatic cells form a continuous mantle—of the branches of the portal vein in the one case, of the branches of the hepatic vein in the other case.

Pervading and filling the spaces left between the arborescent ramifications of the hepatic vein with its cell mantles are the radicles of the portal vein, accompanied by the hepatic artery and the bile-ducts, all supported by connective tissue (the capsule of Glisson), sparse in some places, more abundant in others. But such is the arrangement of the intertwinings of the "portal tree" and the "hepatic tree," that the finest ramifications of the one are everywhere separated from those of the other by a collection (mantle) of liver cells.

As distinguished from most other organs, the liver has two sources of blood supply—the portal vein and the hepatic artery—which commingling form a plexiform capillary network, in the meshes of which the liver cells are arranged. Ultimately, the capillaries unite again to form the radicles of the hepatic vein—the rectilinear veins of Sabourin which empty into the so-called central veins. The circulation within the liver, which is not only interesting in itself, but of much importance in connection with certain features of cirrhosis, has recently been studied attentively by Opie.[1] It has long been known, and Opie directs renewed attention to the fact that one of the important characteristics of the liver is its susceptibility to variations in blood pressure, saying: "The portal vein, hepatic artery, and hepatic vein are in such free communication that the entire lobule can be injected from any one of these vessels. Hence, artificial injections do not define the intralobular distribution of the portal and arterial blood, for doubtless differences of venous and arterial pressure have a part in determining peculiarities of the circulation within the lobule." Opie's experimental studies led him to the conclusion "that both the hepatic artery and portal vein pour their blood into the periphery of the lobule, and here the

[1] Zonal Necrosis of the Liver, Journal of Medical Research, 1901, vol. xii. p. 147.

influence of the arterial blood pressure is most strongly felt. When foreign material reaches the liver by the portal vein it is washed from the peripheral zone by the arterial blood, and tends to be deposited in a middle zone, where the influence of the opposing circulation is less strongly felt. Hence, it is not improbable that toxic substances capable of causing necrosis of the hepatic cells, brought to the liver by the portal vein, might first exert their effect upon cells within a midlobular zone. The periphery of the lobule, moreover, is supplied with arterial blood, and is perhaps for this reason less susceptible to injurious agencies."

CHANGES IN THE PARENCHYMA OF THE LIVER. If we examine attentively the parenchyma of the liver in a moderately advanced or well-advanced case of cirrhosis certain noteworthy deviations from the normal become apparent: (*a*) Changes in the size of the lobules; (*b*) changes in the shape and configuration of the lobules; (*c*) changes in the arrangement of the liver cells; (*d*) degenerative or atrophic and hyperplastic changes in the liver cells; and (*e*) changes in the vascular arrangement and supply.

Changes in the Size of the Lobules. One of the patent and noteworthy features of the lesions of cirrhosis is the marked variation in size of the "granules" or "hobnails," which not only vary considerably in size in different cases, but also in the same case in different parts of the liver, some being notably smaller, others notably larger than the lobules of the normal liver. Long identified as the analogue of the normal liver lobule, it is now well known that many of them are made up of several lobules or of the remains of several lobules; but it is important to bear in mind that some of them are really single lobules, departed, however, somewhat from the normal. The question of the size of these lobules has recently been studied attentively by Kretz, MacCallum, etc. Kretz, pointing out that the normal lobules are roundish-polygonal in shape, with their long axis in the direction of the central vein, states (as is well known) that their greatest diameter is usually a little more than 1.5 mm., and may reach 2.5 mm., whereas the short diameter is about 1 mm., and that in cirrhosis these figures are surpassed, some lobules being much larger, and other collections of cells much smaller. MacCallum found that while the average radius of a lobule (that is, the thickness of the mantle of cells about the central vein) in a number of normal livers is about 0.66 mm., in advanced cirrhosis lobules are encountered that measure from central vein to periphery 1 mm. or more, the small diameter therefore being more than 2 mm. My own observations amply confirm the truth of these statements. I have not, however, been able to confirm Brissaud and Dopter's[1] statement that the lobules of the right lobe are considerably larger than those of the left.

[1] Note sur les differences de volume des lobules hepatiques du foie humain, Gaz hebd de méd, 1902, tome xlix p 661.

Changes in the Shape of the Lobules. With the central vein as a point of departure, we find that, as already stated, the lobules are roundish-polygonal in shape, their long axis being in the direction of the central vein, and their cross-sections roughly rounded, and that the veins are in reality central—that is, surrounded on all sides by an even thickness or mantle of cells. In diagonal or tangential sections of lobules this is not so apparent, and the picture is altered where several lobules join, or where the liver cells vary somewhat their course to form the continuous mantle, especially about the junctions of sublobular and lobular veins. In cirrhosis there is a notable departure from this shape and configuration; many of the lobules are no longer rounded, but quite irregular in transverse section; many of the cell mantles no longer surround the central veins equally on all sides, but very unequally, so that many central veins are situated eccentrically in a more or less misshapen lobule; many so-called lobules have no central vein at all (and are not tangential sections of otherwise normally arranged lobules); certain lobules (or so-called lobules, usually remains of several) have two, three, four, or more central veins; and often the central vein may be seen in the adjacent interlobular connective tissue. Thus, we ascertain that the mantle of cells covering a central vein varies much in thickness in different regions (transverse planes), sometimes exposing, sometimes covering, the vein to a greater or less extent; and that though isolated collections of liver cells (that is, collections devoid of central veins) do occur, some supposedly isolated collections are merely sections of more or less irregularly proliferating dendritic branches of what may be called the parent stem, or mantle covering a single central vein.

Changes in the Arrangement of the Liver Cells. A conspicuous feature of the normal liver lobule is the radial arrangement of the liver cells about the central vein. In cirrhosis, although this arrangement is preserved to some extent, in many places its partial or complete absence is noteworthy. The architecture of the lobule has become altered so that the cells are arranged irregularly, in parallel rows, etc. This rearrangement of the liver cells is quite as well marked in many of the lobules that have a central vein as in those collections of cells entirely devoid of central veins.

Degenerative or Atrophic and Hyperplastic Changes in the Liver Cells. The degenerative and atrophic changes in the liver cells in cirrhosis—simple atrophy, parenchymatous degeneration, fatty degeneration, fatty infiltration, pigmentary infiltration, etc.—are a part of common knowledge and for a moment need not be further discussed. It is important, however, with a view to the correct interpretation of the lesions, to bear in mind that many cells at the periphery of lobules (or so-called "hobnails") in immediate contiguity to wide bands of connective tissue are by no means atrophic, even in fairly well-advanced cases; on the contrary, it is really note-

worthy that whereas *a priori*, in conformity with prevailing opinions, compression atrophy of these cells should be expected, and is, indeed, often commented upon, the cells that should exhibit this alteration most markedly—that is, those of the periphery of the lobule—often present an unusually plump and healthy appearance, and cannot therefore have been unduly compressed.

Special attention, however, should be directed to the evidences of hyperplasia of the liver cells that are readily found in almost every cirrhotic liver, even in far-advanced cases. Mitoses, it is true, are rarely encountered in advanced cases—largely in consequence of the nature of the process, the advanced stage at which the examination is generally undertaken, and the length of time after death that the liver is obtained, time sufficient for the completion of any mitoses present at death. That many of the liver cells, however, are newly formed is quite apparent from their large, clear, and plump appearance, their rounded (rather than polygonal) outline, the presence of two or more nuclei, and the absence of fat and of pigment from their protoplasm. And what is especially noteworthy is that these cells, sometimes small collections of them, are situated amidst other liver cells normal in size and outline, provided with only a single nucleus each (though sometimes with two nuclei, which is quite normal), and the protoplasm of which is normally pigmented and contains fat. These newly formed cells are usually found at the periphery of a lobule; sometimes they make up a larger or smaller sector of a lobule; sometimes they seem to have compressed the older cells of their immediate vicinity; and sometimes small collections of them are found completely surrounded by wide bands of connective tissue, where, by their generally plump and healthy appearance they convict the connective tissue of exerting less constrictive force than they themselves expansive force. The sector of a lobule sometimes made up of these cells serves, in some cases, to restore more or less completely the normal outline of the lobule—that is, of the mantle of cells covering a central vein—in which event a part of the mantle of cells consists of such newly formed cells arranged irregularly, and a part of older cells arranged radially; in other cases this cellular hyperplasia has occurred in such a manner that the central vein is more or less exposed on one side, and on the other covered with a mantle of cells often twice or thrice the normal thickness.

In acute and subacute, or what may be called recurring, cases mitoses are occasionally encountered (doubtless they would be more frequently encountered were our methods of investigation better thereto adapted), and necrotic and hyperplastic changes may be found side by side in the one lobule.

A characteristic feature of almost all cases of cirrhosis, more conspicuous, however, in some cases than in others, is the bile-ducts or so-called pseudobiliary canaliculi, the nature and origin of which

has for a long time occasioned much discussion. Doubtless the great increase of these structures in cirrhosis, as contrasted with the normal liver, is relative only, and due in part, but only in small part, to mutual approximation of already existing bile-ducts occurring in consequence of loss of the liver tissue; but that most of them are newly formed is generally conceded, and is quite apparent from their excessive number and the evidences of active proliferation (mitosis) that they commonly exhibit. Their origin has not been determined definitely for all cases. Dispensing, for the time being, with a detailed discussion of the question, it seems quite certain that some, at least, of them result from proliferation of previously existing bile-ducts; on the other hand, since they are found in a wide variety of disorders, such as all forms of cirrhosis, acute yellow atrophy, in the neighborhood of hydatid cysts, tubercles, gummas, etc., in other words, in diseases alike only in that they destroy liver tissue; since they differ from normal bile-ducts in the absence or relative paucity of encircling elastic tissue; since they have been found by Ackermann and others to be directly continuous with liver cells; and since in cases in which the liver cells have been much destroyed, they are found in large numbers in what may be interpreted as the remains of the skeleton of the lobule, we are quite warranted in the opinion that they may result from proliferation of the liver cells themselves, constituting in this event a reversion to a less highly specialized type of structure. The important fact only that I wish to point out in this connection is the histogenetic equivalence of the liver cells and the epithelium of the bile-ducts, and the fact that proliferative changes of the liver parenchyma is a conspicuous feature of common cirrhosis.

Changes in the Vascular Arrangement and Supply. Reference has already been made to the fact that the central veins are situated eccentrically in certain more or less distorted and rearranged lobules; that they may be found at the edge of certain lobules or remains of lobules, and even in the interlobular connective tissue, and that certain islets of liver tissue are totally devoid of central veins. Another noteworthy feature is that the central veins appear to have diminished in number in the liver as a whole. Corresponding to the smaller size of many of the cirrhotic livers and the associated loss of the liver parenchyma, the central veins in certain regions sometimes exhibit a mutual approximation, which, however, varies much in different livers and in the same liver in different places. In some regions where considerable hepatic-cell hyperplasia has occurred they are often quite widely separated. But were the central veins not diminished in number in the liver as a whole, we should expect to find, in a small so-called atrophic cirrhotic liver a relative increase in the number of central veins in a given square surface as compared with a square surface of similar size in a normal liver—which, however, is not the case. The absolute lessening of the number of central veins becomes further evident

when we recall the many islets of liver tissue (likely at first sight to be interpreted as more or less distorted lobules) entirely devoid of central veins.

Confirming the view that a number of the central veins have disappeared, we may sometimes find in sections happily cut through the desired plane bulbous and distorted side branches of the sublobular and lobular veins. Furthermore, Kretz, who has studied attentively the circulation in the cirrhotic liver, seems conclusively to have proved, by means of injected and macerated preparations, that the finer ramifications of the hepatic vein are fewer than in the normal liver; that the sublobular and lobular branches are more tortuous and in some places larger and thicker, and that they have attached to them bulbous and distorted side branches, representing, according to him, the atrophic remains of central, sublobular, and lobular veins persisting after the disappearance of the related parenchyma.

Apropos of the circulation in cirrhosis Kretz states: "If in ordinary atrophic cirrhosis an injection is made from the portal vein a small part of the hepatic tissue is, as a rule, so easily permeated that the injection mass passes over into the hepatic vein at a time when a very considerable part of the parenchyma is still uninjected; more particularly after such injection experiments the large turgescent islets of tissue and the small areas devoid of hepatic veins . . . remain unaffected. By injecting the hepatic artery, always decidedly enlarged in these cases, these very areas are most easily injected. They may also be partly injected from the portal vein if an injection mass, which passes with difficulty, is employed. Based upon these observations, the circulation in the diseased liver must be depicted as follows: a small part of the portal blood passes through the remnants of the old acinous channels into the hepatic veins; this passage forms an internal portocaval anastomosis supplementing the external; to the remaining branches of the portal vein more than the normal amount of arterial blood is furnished; this, on the one hand, supplies the granular areas of the parenchyma devoid of hepatic veins, and on the other, the hypertrophic remains of acinous tissue. This excess of blood passes under increased pressure through the remainder of the capillaries, narrowed by atrophy of the acini and lengthened by hypertrophy of the liver tissue. The lengthening and narrowing of the capillary vessels and the excess of arterial blood are the true causes of the increase of portal pressure, as is shown by the occurrence of ascites in subacute atrophy of the liver with regeneration, but without contracting connective tissue."

Miura[1] also has made some interesting observations on the changes in the vascular arrangement that occur in cirrhosis.

[1] Beiträge zur Pathologie der Lebercirrhose, Mittheilungen aus der Medicinischen Facultät der Kaiserlich-Japanischen Universität zu Tokio, 1900, Bd. iv. p. 49.

Pointing out that many of the portal radicles (as is well known) gradually become obliterated, he describes the development, within more or less well-preserved lobules, of *communicating vascular* twigs directly uniting the portal veins with the central veins. He terms these internal collateral branches, in contradistinction to the external collaterals (those outside of the liver); probably they are not diverse from, or at least originate from, the rectilinear veins of Sabourin. These internal collateral branches, together with their ramifications and the associated portal and central veins, sometimes become provided with connective tissue and serve to subdivide the lobule—in which event the central veins, situated where trabeculæ of such connective tissue join, are likely to be mistaken for portal veins. Usually, however, they may be distinguished by their comparative paucity of elastic tissue, as contrasted with the portal veins. It is scarcely necessary to discuss in detail the well-known changes in the portal vein itself, its increase in thickness and tortuosity, and the obliteration of many of its radicles, especially in advanced cases.

Changes in the Connective Tissue. A study of the connective tissue in almost any well-marked cirrhotic ("coarsely granular") liver shows that it is made up of several sorts of tissue: (1) Some of it is unquestionably newly formed; of this there is no doubt, the debatable point being as to its nature and genesis; (2) some of it is the tissue originally between the lobules—the connective tissue of Glisson's capsule; and (3) some of it is the collapsed and otherwise altered reticulum of certain liver lobules, the cells of which have disappeared, evidently degenerated by some influence and subsequently removed.

As regards the newly formed tissue, its nature and distribution have been studied by a large number of investigators, and little that is new can be added to what is common knowledge. It has been demonstrated by divers methods of investigation, including the use of Weigert's elastica stain, Mallory's connective-tissue and reticulum stain, digestion with pancreatin, etc., that the normal liver contains three kinds of connective tissue: (1) White fibrous tissue, present in the liver capsule and in large amount in the interlobular spaces; (2) elastic tissue, found chiefly about the bloodvessels, to a less extent about the bile-ducts and in the liver capsule, and to a very slight extent in and about the walls of the central veins; and (3) reticulum; according to Mall[1] "a great share of the tissue accompanying the interlobular vessels and all that about the intralobular is reticulated. The peritoneal surface may be viewed as a thickening of the reticulum, intermixed with some white fibres."

Whereas, formerly the fibrosis of cirrhosis was generally thought

[1] Reticulated Tissue and its Relation to the Connective-tissue Fibrils, Johns Hopkins Hospital Reports, 1896, vol. i. p. 171.

to consist almost, if not quite exclusively, of white fibrous tissue, the studies of Hohenemser,[1] Melnikow-Raswedenko,[2] Flexner,[3] McIntyre,[4] Oliver[5] and others have shown us, and anyone can readily demonstrate, that in reality it consists of all the connective tissues of the normal liver—pathologically altered, it is true. The most conspicuous change is the great increase in the elastic tissue—probably more or less intimately related to the great increase in the capillaries derived from arterioles; this elastic tissue not only makes up a large part of the fibrous bands between the lobules, but it is also found to some extent within the lobules, and occasionally a very small amount of it is found about or in the immediate neighborhood of the central veins. A not inconsiderable part of the fibrosis is made up of reticulum—not only the reticulum normally between the lobules, but also the intralobular reticulum—that remaining after the liver cells have degenerated and been removed. This collapsing intralobular reticulum may be quite readily distinguished in tolerably early cases; later, however, when it has become a part of the interlobular fibrosis it can scarcely be recognized as a distinct entity. I believe that Flexner is quite correct in the opinion that the changes in the reticulum in cirrhosis constitute an hypertrophy rather than an hyperplasia of the fibrils.

The distribution of the connective tissue in cirrhosis has long been the subject of much and more or less fruitless discussion; and the many classifications based thereupon have not tended to clarify any of the obscurity connected with the subject. Multilobular, monolobular, and pericellular (as well as certain more or less synonymous terms, such as annular, circumlobular, unilobular, interlobular, extralobular, intralobular, perilobular, intercellular, etc.) are in common use, and are said to be descriptive of special types of the disease. Without entering upon a detailed discussion of the question at the present time, I may point out that while one or the other of these varieties of distribution of connective tissue may appear to predominate in certain cirrhotic livers, all varieties are much more likely to be encountered in the one liver; and while so-called "hypertrophic" or biliary cirrhosis, as contrasted with so-called "atrophic" cirrhosis, is usually characterized by less interlobular fibrosis and an earlier and a finer intralobular fibrosis, it is frequently impossible from a mere microscopic inspection of a section of a cirrhotic liver

[1] Ueber das Vorkommen von elastischen Fasern bei cirrhotischen Processen der Leber und Niere Virchow's Archiv 1892, Bd cxl p. 192

[2] Histologische Untersuchungen uber das elastische Gewebe in normalen und pathologisch veränderten Organen, Ziegler's Beiträge, 1899, Bd xxvi p 546

[3] The Nature and Distribution of the New Tissue in Cirrhosis of the Liver, University Medical Magazine, 1900–1901, vol xiii p 613

[4] Some Points in the Formation of Connective Tissue in the Liver, Glasgow Medical Journal, 1901, vol lv pp 341, 415

[5] Elastic Tissue in Cirrhosis of the Liver, Transactions of the Chicago Pathological Society, 1901–1903 vol v p 96

to state to which type of the disease it belongs. Assuredly, in the advanced stages the lesions tend to become very much alike. The difficulty is overcome by some writers who postulate so-called "mixed" cases.

THE NATURE OF CIRRHOSIS. Endeavoring to interpret the foregoing lesions, we find that the most noteworthy feature is the complete rearrangement of the architecture of the liver parenchyma. In consequence of some cause or causes larger or smaller portions of numerous liver lobules have been destroyed, many liver lobules have been destroyed entirely—whence the necrotic changes and the skeleton of the reticulum remaining after removal of the cells that are apparent in the early stages, and the eccentric and otherwise irregular situation of many central veins (the variations in size and outline of the cell mantles) and the complete disappearance of other central veins that are apparent in the advanced stages of the disease. Furthermore, evidences of regeneration and hyperplasia of the liver cells are quite apparent—in the general appearance of many of the cells themselves, in their irregular arrangement, in parallel rows, etc. (as contrasted with the normal radial arrangement), in the large hyperplastic lobular (so-called adenomatous) masses, in the irregular dendritic ramifications of liver cells with a peculiar vascularization and entirely devoid of central veins, in the pseudobiliary canaliculi, etc. Although evidences of secondary atrophy of the liver cells are by no means wanting, especially in advanced cases, the peripheral cells of many of the lobules in early cases, but often also in well-advanced cases, exhibit no evidences of compression atrophy whatever, being plump and well formed and manifesting, therefore, more expansive force than the surrounding connective tissue constricting force; some of the small islets of liver tissue containing central veins, since their peripheral cells exhibit no evidences of compression, must have resulted from degeneration and subsequent regeneration of the liver tissue; and some of the small islets devoid of central veins, since they exhibit no evidence of peripheral compression nor of capillary stasis, cannot have resulted from sequestration of a part of a lobule by invading connective tissue, but must represent regenerated liver tissue. The parenchyma of the liver in cirrhosis, then, consists not only of remnants of the original parenchyma, but also and in large part of regenerated liver tissue that differs from normal liver tissue in that it does not reproduce its typical architecture nor its relation to the bloodvessels—the capillaries and the central veins.

That the liver is capable of this really remarkable regeneration has been amply proved—especially by Ponfick,[1] von Podwyssozki,[2]

[1] Experimentelle Beiträge zur Pathologie der Leber, Virchow's Archiv, 1889, Bd. cxviii p. 209; 1890, Bd. cxix. p. 193; 1895, Bd. cxxxviii. (sup. Heft) p. 81.

[2] Experimentelle Untersuchungen über die Regeneration der Drusengewebe, I. Theil, Leber. gewebe, Ziegler's Beiträge, 1886, Bd. i. p. 259.

von Meister,[1] and others, particularly Pearce[2] recently, who have studied regeneration of the liver experimentally; by Marchand,[3] Meder,[4] Stroebe,[5] Barbacci,[6] Mertens,[7] McCallum,[8] and others who have studied the regenerative lesions following acute yellow atrophy; by the many observers (a good collective list of whom is given by Barbacci), who have studied the adenoma formations in the liver; and by Kretz,[9] Greco,[10] MacCallum,[11] and Böstrom,[12] who have studied the regenerative changes in cirrhosis.

The present is not the time to discuss these regenerative changes in detail, and I must content myself with pointing out that the changes found in cirrhosis when compared with those found, for instance, by Ponfick in experimental hypertrophy of the liver, are found to be identical; that is, for a short time after removal of a portion of the liver mitoses are quite abundant, but later the new-formed liver tissue partakes of the characters of the liver cells in cirrhosis that I have already mentioned—large, clear, plump cells with rounded outlines, containing two or more nuclei, and devoid of fat and pigment, and occurring often in the midst of other cells, differing in no way from the common liver cells (smaller, usually polygonal, with a single nucleus, and normally pigmented and containing fat and sometimes even compressed by the newly formed cells).

The nature of the fibrosis in cirrhosis has long engaged the attention of pathologists; whether it is an inflammatory or a replacement fibrosis is still a matter of discussion. Although the question may not be definitely decided offhand, there are many facts which suggest that the prevailing opinion that the cause of cirrhosis, whatever it may be, engenders a primary overgrowth of connective tissue, is not correct. I have just mentioned certain facts that show that the lesions of the parenchyma in cirrhosis cannot with reason be

[1] Ueber die Regeneration der Leberdrüse nach Entfernung ganzer Lappen, Centralbl f. allg. Pathologie und path. Anatomie, 1891, Bd. ii. p 961.

[2] The unpublished paper on Experimental Cirrhosis of the Liver previously referred to.

[3] Ueber Ausgang der acuten Leberatrophie in multiple knotige Hyperplasia, Ziegler's Beiträge, 1895, Bd. xvii. p. 206.

[4] Ueber acute Leberatrophie mit besonderer Berücksichtigung der dabei beobachteten Regenerationserscheinungen, Ziegler's Beitrage, 1895, Bd xvii. p. 143.

[5] Zur Kenntniss der sogenannten acuten Leberatrophie, Ziegler's Beitrage, 1897, Bd. xxi. p. 379.

[6] Ueber Ausgang der acuten Leberatrophie in multiple knotige Hyperplasia, Ziegler's Beitrage, 1901, Bd. xxx. p. 49.

[7] Lesions anatomiques du foie du lapin au cours de l'intoxication chronique par le chloroform et par l'alcohol, Archives international du pharmacodynamie, tome ii. p. 127 (quoted by Kretz)

[8] Regenerative Changes in the Liver after Acute Yellow Atrophy, Johns Hopkins Hospital Reports, 1903, vol. x. p 375.

[9] Loc cit

[10] Ueber Reproduction der Leber bei Cirrhose, Centralbl. f. allg. Pathologie und path. Anatomie, 1898, Bd. ix. p. 371 (abstracted from La Riforma Medica, 1897, vol iii.).

[11] Regenerative Changes in Cirrhosis of the Liver, Journal of the American Medical Association, 1904, vol. xliii. p. 649.

[12] Loc. cit.

regarded as secondary to (dependent upon) changes in the connective tissue, since no connective-tissue change with which we are acquainted is capable of causing the noteworthy rearrangement of the architecture of the liver so characteristic of cirrhosis, and, as a matter of fact, the liver parenchyma, even in many advanced cases, exhibits a notable absence of compression atrophy—a change that is so conspicuous, for instance, about a growing metastatic carcinomatous nodule in the liver.

As is well known, there is a disposition among pathologists during recent years to separate from the purely inflammatory conditions certain fibroses, of which the fibroid heart, the chronic granular and arteriosclerotic kidney, certain so-called senile fibroses, sclerosis of the nervous system, and cirrhosis of the liver are conspicuous examples. The scleroses of the nervous system are now almost universally believed to be examples of replacement fibrosis —hypertrophy and hyperplasia of the neuroglia to replace lost or destroyed nervous elements; in the fibroid heart, in many if not all cases, the cardiac muscle has degenerated in consequence of deficient nutritive supply or other cause and becomes replaced by fibroid connective tissue; and other examples of such replacement fibrosis are found in pseudohypertrophic muscular paralysis and the atrophy of certain fetal structures at or shortly after birth. There is considerable evidence that the fibrosis of cirrhosis also is of the same nature, at least in part. The lesions of acute inflammation are scarcely if ever encountered (in view of the chronicity of the process this excites little comment), and when found they are not infrequently due to some complication; the changes in the reticulum are, as suggested by Flexner, hypertrophic rather than hyperplastic, and may be explained by recourse to Ribbert's theory of tissue tension; the new tissue, as a rule, is unusually rich in intercellular substance, as contrasted with cells; and many of the cells are doubtless only evidences of the effort on the part of nature to remove effete matter—necrotic liver cells. On the other hand, we cannot deny that a poison or other etiological factor capable of causing necrosis of the liver cells may also be capable of setting up inflammatory phenomena in the interstitial tissue, although the parenchymatous cells are probably more susceptible to injurious influences, and in other cases the necrotic liver cells, being foreign bodies, act as a source of irritation, whence it is likely that if the fibrosis is largely a replacement fibrosis in some cases, in other cases it is both a replacement and an inflammatory fibrosis.

As regards the nature of cirrhosis, then, I may say with Kretz that it is a chronic focal or localized recrudescing degeneration and partial destruction of the liver parenchyma attended by marked regenerative phenomena on the part of the liver cells leading to complete transformation of the architecture of the liver and followed by fibrosis. It is not a primary interstitial hepatitis, the

changes in the connective tissue being altogether secondary in importance and sequence, probably in large part a replacement fibrosis to fill up potential lacunæ left by degenerated liver cells, and probably in some cases at least partly inflammatory. In consequence of the activity of some poison, or other etiological factor, larger or smaller areas of the liver tissue, that is, of the cell mantles about the central veins, become necrotic and are removed—whereby the central veins become more or less uncovered, thus on cross-section appearing eccentrically situated in more or less misshapen lobules; in some cases in which the destruction of liver cells has been through the entire radius of the cell mantle the central vein may be entirely exposed and may even be found in the interlobular connective tissue, in which event it is very likely to be mistaken for a branch of the portal vein. The cells destroyed are those about the periphery of the lobules—in the area of portal distribution, conforming to the fact that the etiological factor (poison) is usually carried by the portal vein. In case the poison is carried by the hepatic artery (as seems likely in some general infections) the lesions may be somewhat diverse, in the beginning of the process at least, and it is by no means improbable (though this still awaits proof) that the differences between the common or Laënnec's cirrhosis and so-called hypertrophic cirrhosis are in part at least dependent upon the source (portal vein or hepatic artery), in part also upon the nature, of the provoking cause. Following the primary destruction of certain liver cells, the intact cells, exhibiting a remarkable regenerative capacity, attempt to replace the lost cells and sometimes succeed in restoring more or less completely the volume of the cell mantles. The cells that regenerate are those in the immediate vicinity of those that have become necrotic, that is, in the ordinary case in which the destruction is not widespread, cells toward or at the periphery of the lobule—which thus are not only most exposed to the deleterious influence of poisons carried by the circulation, but in consequence of being better supplied by arterial blood are also better adapted for repair. I am rather inclined to believe, in view of Opie's researches on the circulation within the lobules, that the primary destructive action may be exerted, at least sometimes, in the so-called midlobular zone—at all events, not immediately at the periphery; that the peripheral cells, being better supplied with arterial blood, survive when others nearer the centre of the lobule succumb, and that these peripheral cells also serve as a focus of regeneration, though, of course, the cells nearer the centre of the lobule also regenerate. I am somewhat confirmed in this opinion by the characteristics and disposition of many of the pseudobiliary canaliculi, which are often most numerous in the skeleton of those lobules that have been much destroyed, and seemingly grow from the periphery into the lobule, in a manner quite analogous to Ponfick's and others' descriptions of their growth into granulation tissue after destruction

of the liver cells. As is now generally conceded, these pseudobiliary canaliculi sometimes grow from pre-existing bile-ducts, but as I have already pointed out, and as is well known, they also develop from liver cells, which in this respect are histogenetically equivalent with the epithelium of the bile-ducts. It is also not unlikely that some of the surviving peripheral cells by proliferation give rise to certain of the hyperplastic islets of liver tissue devoid of central veins.

The continuing activity of the etiological factor leads to a continuous or recrudescing death of certain old as well as newly formed liver cells, which in turn is followed by renewed regeneration. This continuous degeneration and regeneration ultimately lead to complete transformation of the architecture of the liver; certain lobules are entirely destroyed, others are partly or completely rearranged, some are markedly hyperplastic though tolerably well arranged, some show dendritic and other irregular proliferation, new islets of liver tissue entirely devoid of central veins appear, and what is especially important, new circulatory conditions—capillary and venous—arise.

Concurrent with the degeneration of the liver cells, the framework of the lobules collapses and participates in the formation of the fibrosis. This fibrosis, formed as previously described, ultimately contracts, and on the one hand causes atrophic changes in the liver cells, and on the other obstruction and obliteration of some of the interlobular and intralobular branches of the portal vein; this latter, though it contributes to, is not the sole cause of, the ascites—other important factors being the aforementioned new circulatory conditions, especially certain noteworthy changes in the hepatic arterial supply (that need not be further discussed here). Ultimately the potentiality for regeneration possessed by the liver cells and manifested to a remarkable degree, not only in the early but also in the late stages, gradually lessens and is finally lost, the general functional capacity of the liver cells being meanwhile compromised by the contracting connective tissue; the degenerative changes predominate over the regenerative, and the patient soon dies, if he has not succumbed earlier to an accidental infection to which his depressed vitality renders him especially susceptible.

The conspicuous change in the cirrhotic liver at necropsy, then, is the evidences of regeneration of the liver parenchyma; the evidences of degeneration, though sufficiently manifest in early cases, in advanced cases are commonly past and represented only by the newly formed and rearranged liver tissue and the fibrosis; and the liver as a whole may be said to represent its, for a time at least, partially successful efforts to compensate the consequences of the continuous or intermittent activity of the etiological factor—usually a poison, though in some cases possibly a substance causing thrombosis of the portal or hepatic artery radicles.

The idea of the primary change in cirrhosis being in the parenchymatous tissue is by no means new. It was entertained many years ago by Frerichs,[1] Ackermann,[2] Orth[3] and others, and, indeed, Laënnec,[4] who originated the term, was not altogether wrong when he looked upon the "granules" as new-growth, though they can scarcely be considered neoplasms, as he believed; the likelihood or possibility of the primary changes being in the parenchymatous tissue is conceded by Ziegler,[5] Rolleston[6] and others; its probability is suggested by the results of the work of the many observers who have studied the subject experimentally; it has recently been insisted upon by Kretz, Böstrom, and MacCallum, and it seems rather conclusively to have been proved recently by Pearce—who, experimenting with hæmagglutinative serums produced primary focal or widespread necrosis (depending upon the dose of the serum employed) with consecutive repair on the part of the liver cells and other changes that much resemble, though they are not identical with, cirrhosis as seen in man.

It behooves us, therefore, to discountenance, as far as possible, the use of the terms atrophic and hypertrophic, since a large liver may be atrophic to a noteworthy degree, and hypertrophy of the liver cells is a conspicuous feature of all small livers. Difficulties in the way of a satisfactory classification, however, are many, of which not the least important is the fact that different etiological factors may cause similar changes in the liver (so-called atrophic cirrhosis with and without antecedent alcoholism), that one factor may give rise to different forms, so-called, of cirrhosis (thus, so-called atrophic cirrhosis, fatty cirrhosis, and hypertrophic cirrhosis may be preceded by definite alcoholism), and that many factors may be operative in the one case. Probably heretofore we have been too much concerned with the size of the liver, which, nevertheless, is a conspicuous clinical feature; but progress is more likely to be achieved by search for the etiological factor than by too much attention to more or less disquieting clinical signs; and we have reason for believing that an organ like the liver which possesses really remarkable inherent capabilities for repair might, although considerably damaged, be able to establish functional sufficiency were the etiological factor, whatever it may be, corrected or removed early —rather earlier, however, than the disease is usually recognized at the present time.

[1] Klinik der Leberkrankheiten, 1861, Bd. ii. p 19, and Atlas (also Translation by the New Sydenham Society)

[2] Ueber hypertrophische und atrophische Lebercirrhose, Virchow's Archiv, 1880, Bd; lxxx. p 396; Die Histogenese und Histologie der Lebercirrhose, Virchow's Archiv, 1889, Bd. cxv. p. 216.

[3] Lehrbuch der spec path Anatomie, 1887, Bd i. p 939

[4] Traité de l'auscultation médiate, 1819, tome i p 368.

[5] Lehrbuch der allg. Pathologie und path. Anatomie, 1898, 9te Aufl, Bd. ii. p 591

[6] Diseases of the Liver, Gall-bladder, and Bile-ducts, 1905, p 196

A CASE OF CONGENITAL HEPATOPTOSIS, SHOWING A MESOHEPAR.*

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The condition recognized as hepatoptosis is one of some rarity about which much has been written. It was first described in a postmortem examination by Heister[1] in 1754, but his article has been overlooked until recently, the credit generally being given to Cantani,[2] an Italian, who reported a case in 1866. Since his time the literature has been searched and collections of cases made by several authors, among whom may be mentioned Wickham Legg,[3] in 1878, with 20 cases; Faure,[4] in 1892, with 54 cases; Graham,[5] of Toronto, in 1895, with 68 cases, and, finally, Ssaweljew,[6] in 1903, who brings the subject practically up to date with 117 cases. Exhaustive discussions are numerous, the theses of Baget,[7] Duverney,[8] Haenselt,[9] and Pellet,[10] with the recent article of Teleky,[11] being the most complete.

By the term hepatoptosis, or floating liver, is meant a complete dislocation of the liver from its position under the diaphragm, and it does not include cases of floating lobe in which a portion of the organ remains in its normal position. The condition must be a primary affection of the liver, and not the result of intrathoracic or subphrenic abscess or tumor.

Taking this definition as our premise, we find 118 cases of complete hepatoptosis on record, 117 of which are tabulated by Ssaweljew, and 1 reported since. As to the pathogenesis of the condition, leaving out the theory of Heister,[1] promulgated in 1754, that it was due to the will of God to prove His omnipotent power, it narrows down to elongation of the ligaments, stretching of the vena cava, and congenital malformation. The condition is prone to occur in any state of laxity of the abdominal walls and decrease of the intra-abdominal tension. The direct causes given are: sex, repeated pregnancies, especially associated with a pendulous abdomen, removal of tumors, tapping of ascites, trauma, overstrain, and tight lacing. An analysis of Ssaweljew's table shows that of his 117 cases 13 occurred in men, 103 in women, and 1 in a child. Of the women 93 were married and 10 single.

* Read before the Clinical and Pathological Section of the Cleveland Academy of Medicine, March 3, 1905.

One point in the etiology of hepatoptosis which has given rise to acrimonious discussion is the occurrence of a mesohepar. Meissner,[12] in 1869, reasoning from an analogy to floating spleen and kidney, strongly advocated on purely theoretical grounds the apposition of the two peritoneal reflections from the liver to the diaphragm, together with their lengthening as the chief factor in the etiology. His cases did not come to autopsy. Leopold,[13] in 1874, and Sutuguin,[14] in 1875, accepted Meissner's theory, but advanced nothing to prove it. In 1885 Landau,[15] basing his statement on the negative results of a large number of postmortem examinations, particularly in infants, denied the occurrence of such a structure. Faure, in his classic monograph, published in 1892, dubs Meissner's theory a pure hypothesis, and insists that it be not considered. Later writers seem to have unanimously accepted Faure's ideas.

Malformations of the liver of any grade, excepting in monsters, are rare. Numerous cases are reported in which the anomalies consist in absence of one or more ligaments, multiple lobulation, hypertrophy, atrophy, or absence of various lobes, accessory lobes, or faulty position due to rotation about an axis. More marked defects are associated with umbilical or diaphragmatic hernia.

The case we report, Louisa L., a native-born American woman, aged thirty-four years, and unmarried, was admitted to Lakeside Hospital (medical number 3854) on November 4, 1903, on the service of Dr. Powell, to whom we wish to express our thanks for permission to report the case.

The patient was admitted for pain and swelling in the right knee and ankle. In the family history the mother died of "liver trouble," and one sister of dropsy, following rheumatism. The family history beyond this was unimportant. In childhood she had had measles, mumps, and chickenpox. There was no history of pulmonary, cardiac, or gastric trouble. The appetite was good. She had always been somewhat constipated. The menstruation began at thirteen years, and had always been regular, though scanty, until three months before admission, when it stopped. She had never been pregnant. She admitted leucorrhœa for the past three or four years, but indignantly denied the possibility of venereal infection. The condition for which the patient was admitted, a subacute rheumatism, has no bearing on the present report. She had never had a serious accident or fall, and said she did not use tight corsets. Nine years before admission she first noticed a tumor in the right side of her abdomen, about the size of her fist. This did not increase in size for two years and gave her no trouble. Since then she thinks it has increased slightly and caused some dragging pain. Three years before admission an exploratory laparotomy was made by a well-known Cleveland surgeon, who diagnosed "sarcoma of the liver (?)" and backed out. Since then the mass has given her no

FIG. 1

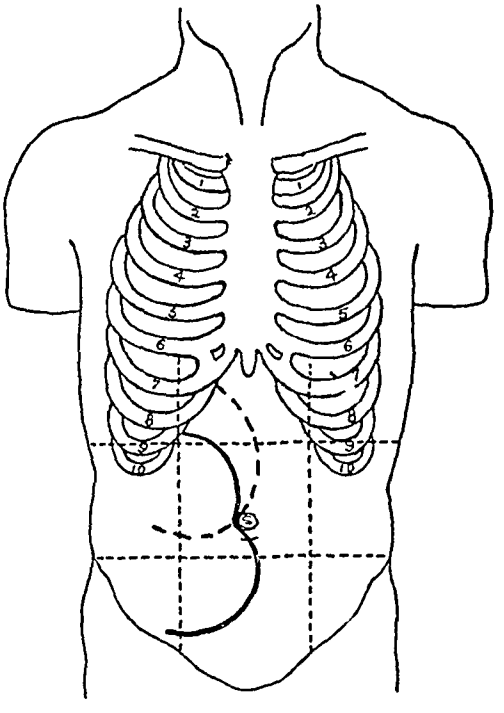


FIG. 1.—Palpation; patient on back. Black = position normally. Dotted = position with liver pushed up.

FIG. 2

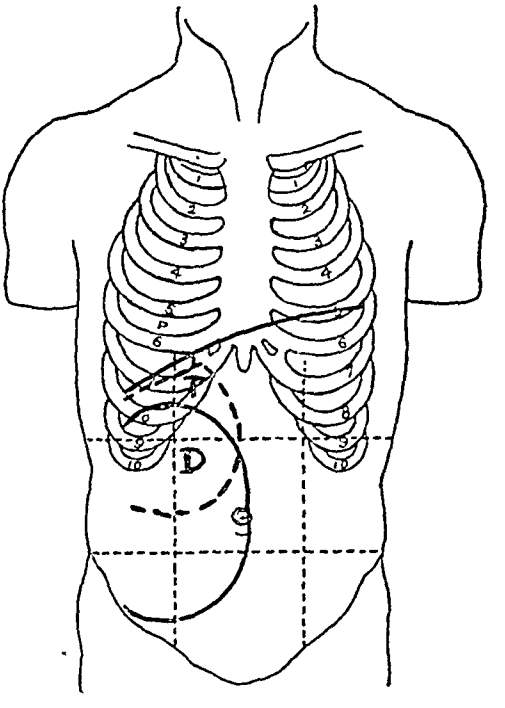


FIG. 2.—Percussion; patient on back. Black = natural position. Dotted = liver pushed up. P = pulmonary resonance. D = dulness. T = area of tympany between P and D.

FIG. 3

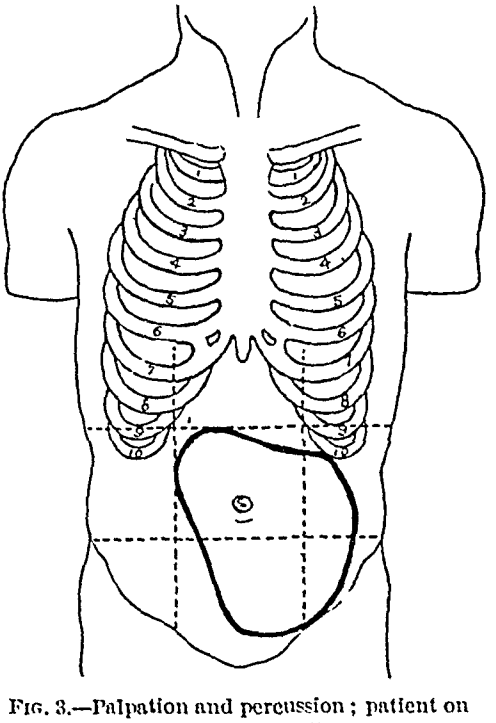


FIG. 3.—Palpation and percussion; patient on left side; position of liver.

FIG. 4

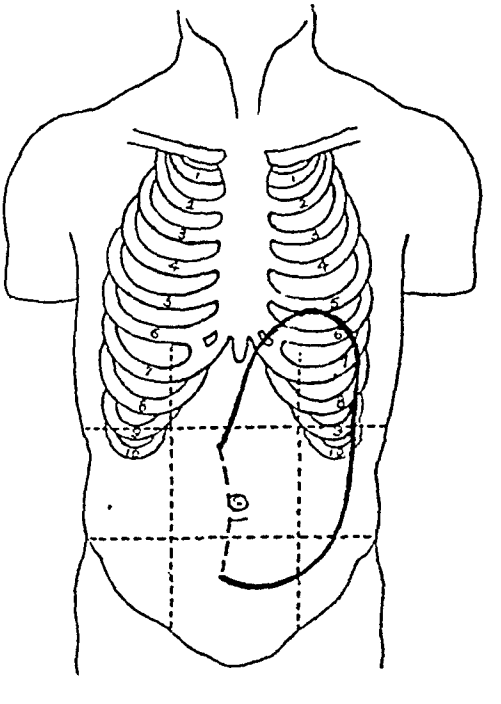


FIG. 4.—Gastric tympany; patient on back.

trouble, except for dragging pains at times on walking. The digestion has been good.

On examination the patient is a well-nourished woman of good color. The conjunctivæ are slightly muddy, but not icteroid. The eyes are normal. The tongue is clean and moist. The teeth are in good condition. There are numerous small areas of brown pigmentation on the cheek, neck, and forearm. The thyroid is slightly enlarged and the right lobe nodular. There is no glandular enlargement. The heart and lungs are normal. The pulse is 72 per minute. The vessel wall is easily palpable.

Abdomen. The abdomen is markedly asymmetrical. There is a definite bulging in the right flank, extending from the costal border to the iliac crest. It reaches to within 2 cm. of the midline just above the navel and to the midline just below it. The mass looks smooth, except for a slight groove running directly laterally from the navel. The most prominent point is 5 cm. to the right and 2.5 cm. below the navel. The mass moves on respiration; no pulsation is seen; there is no visible peristalsis. On palpation the left side of the abdomen is soft and there are no masses felt. The left kidney is just palpable. The spleen is not felt. In the right side of the abdomen (the patient lying on her back) is felt a large, smooth, elastic mass, extending from the midline to the extreme flank. This is easily movable and can be raised by pressure in the right flank. Along the midline of the abdomen it has a definite, sharp, overhanging edge, in which, just to the right of the navel, is a distinct notch. A similar edge can be felt along the lower border. This sharp edge runs from the right anterior superior spine of the ilium in a curved line to the costal border, 4.5 cm. to the right of the midline. The mass extends in the midline 7 cm. below the navel, in the right mammary line 16 cm., and in the anterior axillary line 12 cm. below the costal border, or just to the right anterior spine. It is easily movable and can be forced to any portion of the abdomen. By pressure it can be forced upward 5.5 cm., the upper border disappearing under the costal margin. On percussion the note is flat over the tumor. In the chest pulmonary resonance reaches the seventh costal cartilage in the right parasternal line, the sixth interspace in the right mammary line, the eighth rib in the anterior axillary line, and the ninth rib in the posterior axillary line. At this line the pulmonary resonance changes abruptly to abdominal tympany. There is no liver dulness. The area of tympany extends in the right mammary line 3 cm., and is then replaced by a flat note continuous with the dull area over the tumor. When the liver is forced upward this abnormal area of tympany disappears and is replaced by dulness, which reaches from the right parasternal line to the right flank and comes directly in contact with the area of pulmonary resonance.

By placing the patient on the left side the mass falls to the left

and leaves the right costal border. It then extends from the left Poupart's ligament to a point 2 cm. below the right costal border, a total distance of 24 cm., and has a width at the navel of 18 cm. In this position the left border can be easily felt in the left iliac fossa, and the sharp edge can be picked up between the thumb and finger. In this position the right border can be felt 9 cm. to the right of the navel, and is round, firm, and obtuse. From the upper right border of the mass there is felt a resistance running under the right costal border, resembling a ligament or pedicle, connecting the mass with the structures of the right hypochondrium.

On dilating the stomach by means of sodium bicarbonate and tartaric acid a prominence appears in the lower abdomen, most marked 4.5 cm. to the left and below the navel. The stomach tympany elicited by percussion and auscultatory percussion begins at the fifth interspace in the left parasternal line and extends throughout the left abdomen to a point 8 cm. below the navel in the midline, and to within 4 cm. of the left anterior superior spine of the ilium. To the right of the midline the area of tympany is encroached upon by the flatness of the tumor mass. The right kidney is indefinitely palpable in its normal position.

Vaginal examination shows a small retroflexed uterus, and no connection between the pelvic organs and the tumor mass.

There is marked swelling and tenderness of the right knee and ankle. The right heel is prominent, hard, and sensitive. The shins are clear; there is no œdema. The knee-jerks are normal.

Examination of the blood and urine shows nothing abnormal.

The patient received treatment for her joint trouble from November 4, 1903, to January 23, 1904, with marked improvement. Nothing was done for the liver condition, as it was giving her no trouble, until January 18th, when, on the possibility of the tumor being a gumma, potassium iodide was started in increasing doses. The temperature, pulse, and respiration all remained normal until January 24th, when, with a complaint of pain over the tumor, the temperature rose to 101° and the pulse to 100. The pain increased markedly during the next four days; the temperature rose to 104°; the pulse to 124, and the respirations to 130. An exploratory operation was performed by Dr. Dudley P. Allen on January 29th, and an acute appendicitis found.

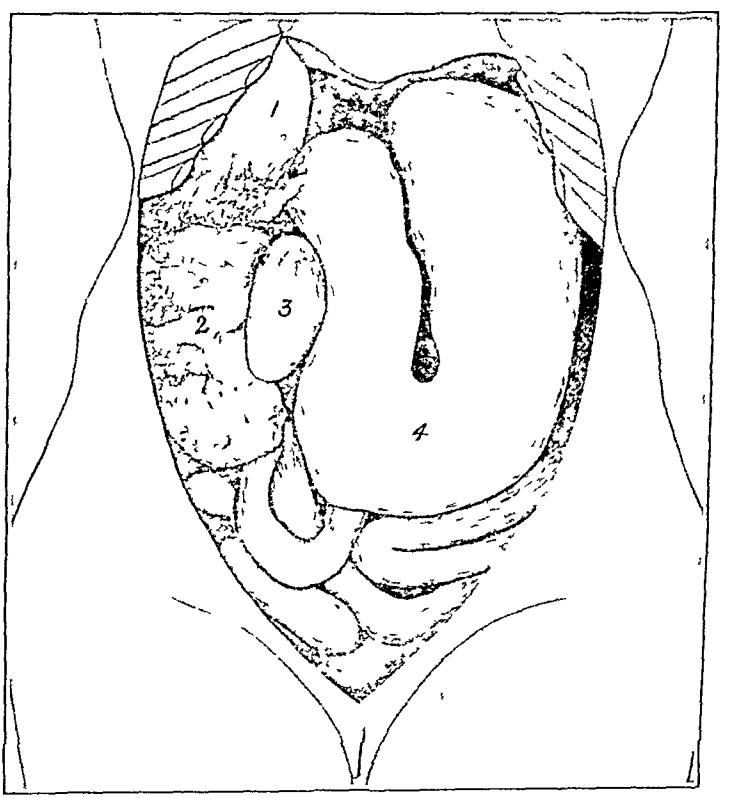
The patient died February 4, 1904. An autopsy was performed the same day.

The point of special interest in the clinical history of this case is the total absence of the accepted etiological factors in floating liver. The patient had never been pregnant, had had no accident, and had not been a heavy worker. Her abdominal walls were not especially lax. The etiological factor could not be determined during life, and the autopsy findings were required to demonstrate it as a case of congenital hepatoptosis.

Pathological Report. The following extracts are made from the autopsy protocol. The autopsy was performed two hours after death.) The body is that of an emaciated white female, 158 cm. long, with beginning *rigor mortis.*

The liver is displaced, lying entirely on the right side. It has two lobes—an upper, corresponding to the left, and a lower, corresponding to the right. Each lobe has a flattened and blunt, ovoid shape, the lower being nearly double the size of the upper. It

FIG 5

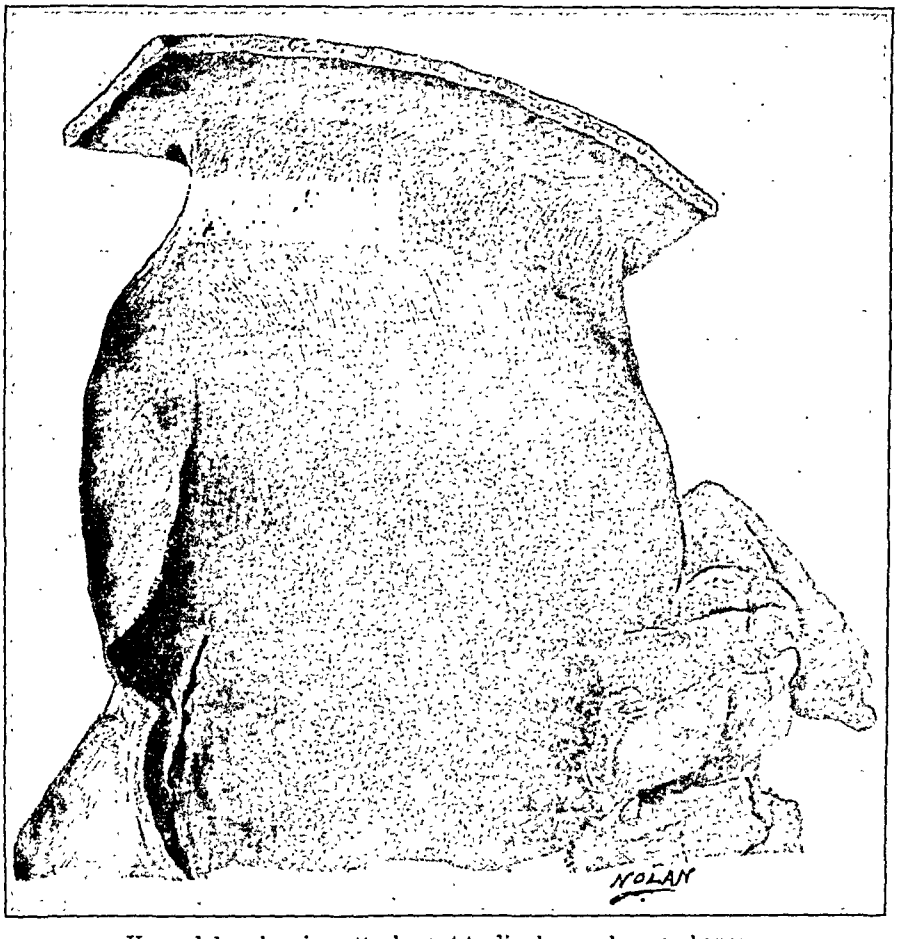


Abdomen at autopsy 1, upper (left) lobe of liver, 2, lower (right) lobe of liver; 3, gall-bladder, 4, stomach

extends 9.5 cm. below the xiphoid cartilage, 8 cm. below the costal margin in the mammary line, and 5.5 cm. in the anterior axillary line. The two lobes are united by an isthmus of dense fibrous tissue, which varies in width from 1.5 cm. on the right side to 3 cm. on the left, and averages 5 mm. in thickness. Scattered throughout this isthmus are isolated liver lobules, some of which are fatty. The lower lobe possesses no ligamentous attachment. The right lateral and coronary ligaments are entirely lacking. Limited to

the upper margin of the upper lobe there is a double reflection of peritoneum to the diaphragm. The liver itself does not touch the diaphragm, and the mesohepar thus formed is 13 mm. in length. The two peritoneal reflections are united only at their free edges, enclosing a space 1 cm. in width at the surface of the liver and a few millimetres at the diaphragm, which is filled with lax fibrous tissue. From the upper half of the right margin of the upper lobe, along the natural line of reflection of the left lateral ligament, there extend

FIG. 6



Upper lobe, showing attachment to diaphragm by mesohepar.

lax and easily torn fibres to the diaphragm. These are regarded as adhesions. The suspensory ligament, instead of being attached along the longitudinal fissure, touches the interlobar isthmus only at its left margin, but it is also attached along the left border of the upper lobe. The round ligament also joins the interlobar isthmus at its left margin. A thick zone of fibrous and fatty tissue, with isolated nodes of liver parenchyma, lies posterior to the junction of the suspensory ligament with the upper lobe. The hepatorenal fold of peritoneum joins the liver at the interlobar isthmus.

The fissure of the gall-bladder appears as an ovoid depression, 4 cm. long, in its normal position with reference to the lower lobe, but its long axis coincides with the long axis of the lobe. The transverse fissure is represented by a deep notch in the upper posterior margin of the lower lobe, from which two fissures branch at an acute angle. The left branch, which is 4 cm. long, is parallel to the gall-bladder fissure and 1.5 cm. from it. The right branch, which is longer, is poorly defined at its origin, but deep at its extremiyt. The subdivisions of the right branch of the portal enter these fissures. The hepatic ducts leave the liver and the hepatic artery and the portal vein enter it at the left margin of the interlobar isthmus. The artery divides 4 cm. and the vein 5 cm. before entering the liver. The relative position of these vessels is normal. The fissure of the vena cava crosses the left posterior upper extremity of the upper lobe. The umbilical fissure and the fissure of the ductus venosus are lost in the separation of the lobes. The quadrate lobe is probably represented by the portion between the gall-bladder fissure and the left branch of the transverse fissure. The caudate and Spigelian lobes have necessarily no analogues.

The liver weighs 1385 grams. The upper lobe measures 13.8x 10.8 x 6 cm., the lower 17.5 x 11.5 x 8 cm. The liver capsule is moderately thickened. The surface of the lower lobe is finely nodular, but that of the upper is more smooth. The lower lobe cuts with greatly increased resistance, and its cut surface is granular. The lobules vary much in size. There is a distinct increase of interlobular fibrous tissue. The liver tissue is of a dull brownish-yellow color. Over the surface and throughout the organ are scattered minute bright-yellow areas. The upper lobe has a smooth surface on section, its resistance is but slightly increased, and the tissue is flabby in consistence. Occupying the centre of the upper lobe is an irregular mass of mixed pearly and translucent fibrous tissue, having the shape of a three-pointed star. One point extends nearly to the left margin, while the other two extend only half-way to the right. Throughout the mass are scattered irregular nodules, varying in size from a few millimetres to 1 cm. in diameter. These project above the cut surface and consist of a firm but friable and structureless tissue of a homogeneous yellow color.

The gall-bladder, which is much enlarged, is U-shaped, and lies between the stomach and the lower lobe of the liver. The distal arm, which is more distended, lies anteriorly. It is 9 cm. long and 5 cm. in diameter. The proximal arm is the same length, but gradually narrows to the orifice of the cystic duct. The organ is not adherent.

The stomach also has a U-shape and is enormously dilated. It extends 11 cm. below the xiphoid cartilage. The cardiac arm of the U measures 22.5 cm. in length and the pyloric arm 16.5 cm. The diameter at the cardiac end is 19 cm., at the middle 9.5 cm., from which the size diminishes gradually to the pylorus. The pylorus is in its normal position.

The appendix is absent and closure of the wound of operation is not perfect, intestinal contents exuding from a minute hole. In the right ileocæcal region the intestinal surfaces are united by fibrinous exudate and have a dark necrotic appearance. Similar exudate is found in moderate amount over the liver and the spleen. There is a scanty amount of brownish, turbid fluid free in the peritoneal cavity. The transverse colon is prolapsed and collapsed, and lies behind both liver and stomach.

The mucosa of an area, with a diameter of 4 to 6 cm. about the orifice of the appendix, is ulcerated. The ulcers have an irregular shape, with a sloping edge and a necrotic floor. The intervening tissue is intensely congested. The mucosa of the rest of the cæcum and colon is smooth, but indurated and mottled with dark-brown pigmentation. The remaining viscera are normal in their positions.

Microscopic. Sections from the new-formed tissue in the upper lobe of the liver show typical gummata. The periportal tissue is especially increased in the lower lobe, with atrophy and necrosis of the liver cells. Periarteritis and endarteritis are well marked.

There are extensive areas of coagulation necrosis and purulent infiltration in the mucosa and submucosa of the cæcum. Some ulcers extend to the submucosa. The acute process is so well marked as to obscure any old lesions, but in the submucosa of the colon are areas of vascular and well-organized granulation tissue, with extensive proliferation of the adventitia of the small arterioles. Some larger arterioles are occluded by a recent endarteritis. An enormous amount of a coarse brownish pigment is deposited in the mucosa of the large intestine, both in cells and free. It does not give an iron reaction.

Pathological Diagnosis. Congenital malformation and displacement of the liver; multiple gummata of the liver, with syphilitic cirrhosis; chronic passive congestion of the portal system; operative removal of the appendix; acute general peritonitis; dilatation of the stomach with atrophic gastritis; acute ulcerative typhlitis; chronic enterocolitis; syphilitic endarteritis and periarteritis of the large intestine; hypertrophy of the thyroid; tuberculosis of the tracheal glands.

We are indebted to Dr. W. T. Howard, Jr., for the use of material and for his supervision of the work.

CONCLUSIONS. 1. The abnormal position of the liver, with its limited mobility, its atypical shape, the absence of the coronary and right lateral ligaments, the abnormal origin of the left lateral ligament, which assumes the function of the coronary ligament, and of the suspensory and round ligaments, the shifting of the fissure of the vena cava from one lobe to the other, though the course of the vessel is normal, together with other variations, leave no doubt as to the embryonic origin of the condition. Syphilis was merely a coincidence.

2. As the review of the literature, particularly of the references given in the *Anatomischer Anzeiger* for the past fifteen years, and of the collections of Jacquemot[16] and Faure,[4] shows no identical case, and but one slightly resembling the present one, it must be considered as unique.

3. The case, though it does not prove that a mesohepar is the cause of hepatoptosis, does prove Meissner's hypothesis that such a condition may exist.

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CHRONIC PARENCHYMATOUS NEPHRITIS; ACUTE GENERAL INFECTION: INFARCTION OF THE LUNG; DOUBLE PHLEBITIS; NEPHROLITHIASIS; PARTIAL RECOVERY.

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GEORGE H., aged thirty-nine years; white; married; consulted me on April 20, 1904, on account of albuminuria. His family history was negative. He had had no sickness of any sort since childhood, except that six weeks previously he had suffered from a pharyngitis, which lasted for a week, and which possibly was of importance, as he had been in contact with a scarlet-fever patient. His throat, however, healed promptly and he felt very well afterward. His habits had always been good; he had never been an alcoholic, although for several years had been accustomed to drink two bottles of domestic beer at night; he had used tobacco in moderation. He lived in Baltimore, but his place of business was in Washington, so that it was necessary for him to get up at 6 o'clock in the morning in order to take the 7 o'clock train for that city, returning in the evening. This mode of life had been followed for eight years,

and he was always in a hurry going in the morning and coming back in the evening. He was in the habit of sitting up late at night, and frequently did not get enough sleep. Shortly before he consulted me he noticed that he became fatigued after slight exertion and that he had shortness of breath. An examination of his urine at that time showed a large amount of albumin; the specific gravity was 1012. The amount passed in the twenty-four hours seemed to be normal, but no accurate record on this last point had been kept. On microscopic examination granular, epithelial, and hyaline casts were found, but not in great numbers.

Physical Examination. Medium-sized, well-nourished man; pallor marked; no œdema of the face or extremities; chest broad, expansion good and equal; lungs clear. *Heart:* Apex in normal position, sounds soft, distant; no accentuation; the bloodvessels are somewhat thickened, but are easily compressed; tension normal. The patient has a remarkable degree of self-control; he is not nervous, and informs me that, except for his weakness and shortness of breath, he never felt better than he does now. Blood count: 3,500,000 reds; leukocytes, 10,000; blood pressure, 140; temperature normal. He was put to bed, but, as the amount of albumin increased and he developed œdema of the legs, he was taken to the Union Protestant Infirmary.

May 2d. The patient complains of a violent pain in the left side of the chest under the eighth rib behind. Over this area there is heard a localized pleural rub, with slight increased conduction of the voice and breath sounds.

4th. The pain has disappeared; the color has improved; 800 c.c. of urine were voided in the last twenty-four hours; specific gravity, 1022; white blood count, 5000; blood negative for malaria; hæmoglobin 90 per cent.

6th. Has had a restless night and feels much worse; his pulse is rapid, and his urine shows more albumin than for several days. Numerous rales are heard throughout the lungs and the aortic second sound is accentuated; the heart's action is rapid and weak. The swelling about the ankles is diminishing.

9th. The patient has complained of a pain in his right side behind. A friction rub, with increased conduction of voice and breath sounds, is present over an area beginning at an imaginary line two inches to the left of the spine and extending from the eighth to the tenth ribs. A few crackling rales are heard at each base.

10th. During the night the left thigh suddenly became swollen and the patient complained of a good deal of pain in the groin. The veins of the left leg are distended and a mass can be felt just below Poupart's ligament; for the last few days the patient has had a slightly elevated temperature.

12th. The whole left leg is much swollen; there is no œdema of the right leg; the veins of the left leg are palpable at the saphenous

opening and the toes are blue; there is swelling of the scrotum and penis; the friction rub noted on the ninth has disappeared. By night the swelling in the leg had increased and muscular contractions in the calf muscles had appeared. The patient looked like a very ill man; his face was pinched; he was much depressed and very weak.

13th. His condition has steadily grown worse; muscular contractions are frequent; in the last twenty-four hours he has passed only 520 c.c. of urine; the penis and scrotum are enormously swollen; the pulse is rapid and of poor volume. The blood count shows 13,800 leukocytes; the blood pressure is 130. Considering his extremely grave condition, Dr. Finney and I decided it would be well to try decapsulation of the kidneys. This was done at 3 P.M. The patient was put to sleep and a strip taken from the capsule of each kidney. The left kidney was slightly larger than normal; the right kidney much larger. Chloroform was employed for anæsthesia. The patient regained consciousness quickly and by midnight is feeling comfortable. There is no pain in the leg; some few rales are heard over the chest; the pulse is full, strong, and regular; the muscular contractions have disappeared, and apparently there has been a slight increase in the amount of urine passed; the general condition seems better.

14th. The patient slept well last night; the pulse is somewhat improved, but the patient is exhausted; he does not complain of pain in the regions of the incision; the left leg is swollen, but does not inconvenience him. The leukocytes have increased to 16,800. During the next few days his general condition improved, but there was no diminution in the œdema, the amount of albumin passed or the number of casts seen on microscopic examination. The wounds in the back healed promptly.

From May 27th the œdema rapidly increased, so that on June 1st the scrotum was tapped in five or six places, with the loss of a large amount of fluid. During this month his condition remained practically about the same, except that his strength improved, and toward the last of the month he was kept out-of-doors.

From July 1st there was a gradual increase in the general œdema, so that by the 16th he was practically waterlogged. On that date his legs were scarified in three places each, with the loss of a great deal of fluid. An attempt was made to tap the abdomen, but without success, as there was no free fluid in the cavity. The draining was very effectual and the legs, scrotum, and penis slowly came back to normal size.

During August his condition was about the same. After the openings in the legs closed the swelling came back, so that by the 16th it was necessary to scarify the legs and scrotum, which again relieved him.

August 27th. The patient began to suffer from an attack of pain and swelling in the right leg, similar to that which he had had in the

left. The veins became prominent and he suffered a great deal for two days.

31*st*. The condition has improved. Liver dulness begins at the lower border of the sixth rib, and the edge is palpable two fingers' breadth below the costal margin; his right leg is swollen, the veins are prominent, and he complains of considerable discomfort.

September 7th. Patient left the hospital to-day; his legs have not been draining for the past three days; there is no œdema about the body and he feels better; the scrotum has diminished in size; the patient eats and sleeps well. Toward the end of the month it was again necessary to scarify the legs, and again the procedure proved beneficial; the œdema went down and the wounds healed, so that by November 4th there was no discharge. Again the œdema increased, and he was tapped on November 10th, with slight improvement. He was at a standstill until November 20th, when he was taken with colicky pains in the region of the right kidney. With the attack there was an increase in the amount of urine, the daily average for the first week being 1400 c.c. The kidney pain was as severe, but paroxysmal. At the end of a week he commenced to pass small calculi, phosphatic in nature, and the pain stopped. On the next the left kidney was attacked and the amount of urine dropped steadily. On the fourth day the pain disappeared and only 30 c.c. of urine were passed. The next day the patient was somnolent, he had no pain, his pulse was rapid, of fairly good volume, and the œdema was increasing. He was bled, eighteen ounces being removed, and in the next twenty-four hours he passed a large amount of urine, with a specific gravity of 1008 and an abundance of albumin. The amount of urine gradually decreased, but the œdema disappeared. In two weeks' time the swelling commenced again and extended up from his feet, involving his whole body and face. The legs were again tapped, with but little effect.

December 14th. General condition not good; swelling of the face and body marked.

15*th*. He was placed on a salt-free diet, and in the next few days there was a remarkable change for the better. The œdema rapidly decreased; the thirst, which before had been intense, left him entirely; the mouth became moist and the general condition improved greatly. After the first week the amount of urine passed fell, but in proportion to the fluid taken was greater than it had ever been, and from that time on the patient made a steady improvement, so far as subjective symptoms were concerned, although the amount of albumin in his urine remained high.

He is now, November 1, 1905, passing a normal amount of urine, with a specific gravity around 1030; 0.3 per cent. albumin; he is actively engaged in business and feels strong and well. Toward evening he has a slight amount of swelling about his ankles, especially in his left leg, which is possibly due to his old phlebitis. He has prac-

tically become a vegetarian, as the taste of meat without salt is most distasteful to him. On two or three occasions he has used salt in his food, and, feeling badly afterward, has determined never to take any more unless the craving for it is irresistible.

During the five months that this man was in the hospital a daily record was kept in which were specified the fluid intake, the amount of urine passed, the amount of nitrogen output, and the percentage of albumin. Since leaving the hospital the total amount of fluid taken and the amount of urine passed have been recorded.

As to the total amount of nitrogen: This varied somewhat, but on the whole did not teach us much. After the Edebohls operation on May 13, 1904, coincident with the rise in the amount of urine, there was a very decided rise in the output of nitrogen, the amount being about double for the first four days that excreted before the operation. At the same time there was a fall in the percentage of albumin, although the total amount of albumin excreted remained the same. All through his attack, however, there was at no time any fall in the amount of nitrogen excreted, except in proportion to the amount taken in, and it is interesting to note that only once was there a threatening of uræmia, namely, after the suppression of urine that came with the attack of kidney stone.

The *albumin* was and has remained persistently high and has been excreted in about the same amount.

Casts were never numerous, but always to be found.

The amount of urine passed in comparison with the amount of fluid taken showed one particularly interesting point. Before the salt-free diet was instituted the amount of fluid taken was on a par with or somewhat in excess of the amount of the urine output; but afterward the intake was only one-half of the output.

The *blood pressure* was never higher than 160, and usually remained around 130 mm.

No accentuation of the aortic second sound has ever been noted.

TREATMENT. For the first five weeks von Noorden's idea as to diet was followed. The milk was modified to contain a low percentage of proteid, and calcium carbonate was given with each portion. From this procedure no apparent result was obtained, the amount of albumin passed in the twenty-four hours remaining constant. As was to be expected, the total amount of nitrogen fell, but only in proportion to the amount of the nitrogen intake. Although there was no betterment in the condition of the kidneys as the result of this diet, it should be remembered that we had, in addition to the chronic condition, an acute infection. After the first five weeks the patient was given broths without meat, a small amount of starchy food, and unmodified milk.

Water. During the first part of the treatment lithia water and distilled water were used in large quantities, but it soon became apparent that the kidneys could not bear over a certain amount

and a restriction was placed upon the fluid intake. It was noted several times that when over a certain amount of water was given the œdema increased and the amount of urine was diminished; whereas after the amount of water was cut down the œdema diminished and the amount of urine output increased.

Drugs. Of the diuretics he was given infusion of digitalis, potassium citrate, tincture of iron, buchu, diuretin, with alkalies and squills. Of these, syrup of squills seemed to have some but only a temporary effect. Half a teaspoonful was given four times a day, and a study of the urine in Table I. before and after its administration shows that there was apparently a decided increase in the amount of urine passed; certainly the œdema was diminished. This drug was used over a period of two weeks, and when it began to lose its effect it was stopped. It caused no nausea and no depression. No results could be directly ascribed to the action of the other drugs; in fact, they seemed to do harm, especially digitalis. Magnesium sulphate was given frequently, and, by acting on the intestine, drained off a certain amount of fluid, but it did not increase the amount of urine passed. Elaterium was used twice, with no good effect, and it caused a good deal of pain. A pill composed of calomel, digitalis, and squills acted badly, the calomel causing an increase in the amount of albumin and a fall in the amount of urine.

The only two drugs that seemed to benefit him at all, then, were squills and the aperients, and it is to be remembered that the squills was used at the same time that the scrotum was tapped. This drug was tried again and had no effect in increasing the amount of urine passed.

In contrast with the effects of the drugs those resulting from what may be called *mechanical agents* were most striking. *Tapping the scrotum* not only relieved the œdema considerably, but a study of the table will show a direct increase in the amount of urine secreted. The same result followed tapping the legs, the resulting drainage being most profuse, except on the last occasion. Scarification of the scrotum was practised twice, the first time being followed by most noteworthy results.

The Edebohls Operation. The result was disappointing in some respects. The amount of urine increased considerably for a few days, but the œdema did not diminish, and during that period the patient was getting a larger amount of fluid than usual. On the day of operation and the one following the patient was given subcutaneous injections of salt solution. It was noticed when these were given that they were absorbed slowly. This fact is easily understood, and the employment of this procedure was certainly a therapeutic error; there was an increase in the amount of urine passed, but the intake of fluids was out of proportion to that amount.

The patient's general condition improved very much after the operation. He lost what he had had before in a marked degree—a

septic look—and his leukocyte count increased considerably. When the capsule of his kidneys was detached there was more pressure than usual. The kidneys welled up out of the wounds as if pressure had been removed.

The patient relapsed into his former state, so far as the condition of the kidneys went, in a very few days, and no sign of kidney betterment could be noted, the amount of albumin being increased. For the first four days after the operation the nitrogen output was doubled, but it is more than probable that this change would have taken place in a normal individual after an operation. The following table shows the amount of urine passed and the amount of fluid taken before decapsulation of the kidneys, while he was taking the squills and after the scrotum was tapped for the first time:

TABLE I.

	Amount of urine passed.	Amount of fluid taken.		Amount of urine passed.	Amount of fluid taken.
May 10	1368 c.c.	3250 c.c.	May 28	760 c.c.	1400 c.c.
" 11	1000 "	1875 "	" 29	1000 "	1800 " [2]
" 12	840 "	2000 "	" 30	1000 "	1800 "
" 13	520 "	2300 " [1]	" 31	1700 "	2000 " [3]
" 14	1510 "	1800 "	June 1	1400 "	1560 "
" 15	1240 "	1500 "	" 2	3130 "	1500 "
" 16	1300 "	1700 "	" 3	1650 "	1510 "
" 17	1300 "	1600 "	" 4	2200 "	1020 "
" 18	1780 "	2000 "	" 5	1570 "	1000 "
" 19	1740 "	1500 "	" 6	1300 "	1250 "
" 20	1030 "	1800 "	" 7	1190 "	750 "
" 21	1290 "	1800 "	" 8	2440 "	800 "
" 22	1410 "	1600 "	" 9	1430 "	490 "
" 23	1450 "	1600 "	" 10	1580 "	1020 "
" 24	1430 "	1600 "	" 11	1560 "	1000 "
" 25	1450 "	1430 "	" 12	1360 "	1000 "
" 26	1020 "	1440 "	" 13	870 "	750 " [4]
" 27	1180 "	1440 "	" 14	800 "	490 "

[1] Operation, decapsulation both kidneys.
[2] Squills.
[3] Scrotum tapped.
[4] Squills stopped.

For increasing diuresis venesection showed the most remarkable results. For the first time in the course of the illness the amount of urine passed was sufficient to drain away all the œdema. In this connection it would seem almost justifiable to think that there might have been the same increase after decapsulation had salt solution not been used; for, after all, venesection acts on an engorged kidney by relieving tension. The effect of venesection was not prolonged, but while it lasted it was certainly very remarkable. As was mentioned above, the procedure was resorted to for almost complete suppression of the urine following an attack of nephrolithiasis, and a study of this chart shows that the mechanical irritation exerted by the stones as long as only one kidney was involved produced a diuresis of considerable amount; but that when the second kidney became affected there was almost complete suppression of the urine, followed by the polyuria after the bloodletting.

TABLE II.

	Amount of urine passed.	Amount of fluid taken.		Amount of urine passed.	Amount of fluid taken.
Oct. 29	570 c.c.	550 c.c.[1]	Nov. 11	330 c.c.	200 c.c.[4]
" 30	570 "	510 "	" 12	180 "	720 "
" 31	730 "	660 "	" 13	30 "	390 " [5]
Nov. 1	1270 "	850 "	" 14	4590 "	1580 "
" 2	1110 "	900 " [2]	" 15	6720 "	2550 "
" 3	1120 "	830 " [3]	" 16	2860 "	1030 "
" 4	1020 "	630 "	" 17	1910 "	1720 "
" 5	2160 "	960 "	" 18	1720 "	580 "
" 6	2370 "	1240 "	" 19	1825 "	820 "
" 7	970 "	670 "	" 20	1540 "	760 "
" 8	1200 "	860 "	" 21	1010 "	700 "
" 9	850 "	805 "	" 22	1230 "	900 "
" 10	460 "	640 "	" 23	1040 "	820 "

[1] Kidney colic, right kidney. [2] Kidney colic. [3] Kidney colic.
[4] Kidney colic, left kidney. [5] Venesection, 18 ounces.

The effect of the *salt-free diet* was interesting for several reasons. The results were more permanent than any obtained before. In the first place, it brought about from its use a reversal of the relation between the amount of fluid intake and the amount of urine excreted; but besides this there were several points that are well worth considering. Before taking this diet the patient had complained continuously of intense thirst, and to allay it was sipping water constantly. After salt was omitted from the food, and this was done absolutely as far as could be, the thirst disappeared, and the tongue and mucous membranes, instead of being dry, as they formerly were, became soft, and there was so marked an absence of thirst that the patient had to be directed to take a certain amount of fluid in order to prevent too heavy a burden upon the crippled kidneys. Moreover, he felt better soon after starting it than at any previous time during his illness, and his betterment has continued. He has found out himself that he is decidedly worse and that the œdema increases as soon as he goes back to salt in his food. As a result he has practically become almost a vegetarian, as he finds meat is very unpalatable without salt.

TABLE III.

	Amount of urine passed.	Amount of fluid taken.		Amount of urine passed.	Amount of fluid taken.
Dec. 10	1250 c.c.	1970 c.c.	Dec. 20	1010 c c.	300 "
" 11	1190 "	1840 "	" 21	730 "	180 "
" 12	1120 "	1450 "	" 22	850 "	360 "
" 13	1230 "	1610 "	" 23	610 "	320 "
" 14	1170 "	1100 " [1]	" 24	771 "	420 "
" 15	2050 "	160 "	" 25	980 "	570 "
" 16	2190 "	160 "	" 26	960 "	410 "
" 17	2440 "	160 "	" 27	950 "	430 "
" 18	1650 "	240 "	" 28	610 "	320 "
" 19	1180 "	150 "	" 29	850 "	610 "

[1] Patient put on salt-free diet.

SUMMARY. *Diagnosis.* Chronic and parenchymatous nephritis; acute general infection resulting in infarction of the lungs; double phlebitis; nephrolithiasis of both kidneys.

Prognosis. On two occasions during the course of the year the patient was manifestly close to death: the first time just before the operation for decapsulation of the kidneys, and the second time after the attack of kidney colic, when there was almost complete suppression of the urine.

It is manifestly impossible to be at all sure about the outcome in this case, but the whole course of the disease shows that there must have been, and must be still, a certain amount of healthy kidney tissue left. Therefore, despite the desperate illness through which the man has passed, and despite the fact that the excretion of albumin and casts has not changed, one may venture to hope that there are an indefinite number of years before this man, during which he may live and possibly still do good work. My experience, especially of late years, has taught me the great uncertainty and wide variations in the course of kidney cases. No general rule can be laid down, but a complete study of the individual equation will sometimes help us in establishing a prognosis that will prove approximately correct.

So far as the treatment in this case is concerned, drugs exerted practically no effect, and the same may be said as regards diet, with the exception that exclusion of salt from all articles of food unquestionably had a great influence upon the œdema.

The Edebohls operation in this case was of doubtful utility. My impression is that it did no good beyond relieving tension at the time.

Venesection acted wonderfully as a diuretic, but produced no permanent result.

A last point, and one which is too often forgotten, is that patients suffering from parenchymatous nephritis are embarrassed rather than benefited by subcutaneous injections of salt solution. In fact, we are to be on our guard about using this procedure, because at times it may prove exceedingly dangerous.

FATAL OBLITERATING ENDOPHLEBITIS OF THE HEPATIC VEINS.

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(From Prof Chiari's Pathologico-anatomical Institute, Prague.)

A DISEASE characterized by a gradual proliferation of the lining wall of the hepatic veins and resulting obstruction, with all its disastrous consequences to the liver and portal system, was men-

tioned for the first time by Budd in 1846 in his treatise on the liver. Since that time numerous similar cases have appeared in the literature, some 23 in all, the majority of which have been published in the last ten years. The German, French, Swiss, Italian, and English literature contains examples of this obliterating endophlebitis of the hepatic veins, but as yet no case has appeared in American scientific or clinical publications. We can hardly believe that cases of this nature have not at some time presented themselves in the clinics of our country. Moreover, when we consider that the outcome of the disease is invariably fatal, the conviction is strengthened that postmortem examinations upon such cases have also been made without the true cause of the pathological lesions and of death being disclosed. This is very easily explained and understood when we realize that, clinically, the cases mask themselves under the every-day garb of a Laennec cirrhosis, and, therefore, are not apt to attract particular consideration; and that at the autopsy, unless the hepatic portion of the inferior vena cava is opened, and the ostia of the hepatic veins there examined, the clinical diagnosis of cirrhosis of the liver may readily be declared to have been correct. To bring out more forcibly the point that this condition is overlooked, not only in the United States but elsewhere, and is by no means as rare as the paucity of reported cases would lead one to believe, let me call attention to the fact that five of the 23 cases below mentioned are reported from one pathological institute, that of Chiari, of Prague, and that since his thorough exposition of the subject, in 1899, two further cases have been found by him at autopsy. It is therefore to bring this condition to the notice of the clinician and pathologist that I publish a full report of a case of this disease. For I feel convinced that we cannot hope to obtain a clearer insight into its cause, or a recognition of its clinical aspects, except by a consideration of many more cases than have been studied and collated up to the present time. In order to give the reader a comprehensive idea of the symptomatology and pathology of a disease which is as yet so little known, I have prepared a tabular synopsis of all the cases which I have been able to find, and shall precede the report of the case which suggested the article, by a general survey of all the data at hand. The facts are in each case taken directly from the original reports, the evident omission of important data being due to lack of information furnished by the author.

Case.	Age and sex.	Clinical data	Autopsy.	Microscopic data.	Conclusions of the author.
Budd, 1846	52 years ♂		Hepatic veins thickened and containing a "pseudomembrane," thrombosis of portal veins; adhesive perihepatitis, pericarditis, and pleuritis		No cause assigned for thickening of veins; the adhesive serositis attributed to irregular mode of life, probably alcoholism being meant.
Cohn, 1860			Widespread phlebitis of hepatic veins; thrombosis of portal veins; ascites, enlarged spleen		
Frerichs (1), 1861	38 years ♀	Abdominal pain, sacculated peritoneal exudate, slight icterus, œdema of lower half of body.	Ostia of hepatic veins closed by complete or partly fenestrated septa and surrounded by thick connective-tissue sheath; liver smaller by a third firmly bound down the capsule thickened, sending firm bands into liver tissue		The valve-like closure of hepatic veins due to a primary peritonitis causing a secondary perihepatitis with connective-tissue bands entering the liver tissue and pressing on the venous walls.
Frerichs (2)	15 years ♀	Ascites, diarrhœa, icterus, enlarged spleen.	One hepatic vein completely closed by a light red mass, the size of a cherry, one ending blind with wall greatly thickened; a third containing adherent thrombi; the vena cava containing round plaques its walls flattened together by connective-tissue bands; loosely attached thrombi in portal vein; liver somewhat small, connective-tissue masses on under surface, extending into parenchyma		Adhesive phlebitis of hepatic veins due to chronic peritonitis.
Rosenblatt, 1867	27 years ♀	Sticking abdominal pain, ascites, distention of abdominal veins	Ostia of hepatic veins entirely absent but represented by slight elevations and depressions of wall of vena cava; left lobe of liver tightly bound down, bands extending from capsule into parenchyma, primary carcinoma of liver with carcinomatous peritonitis		Hepatic veins obstructed as result of fetal hepatitis.
Gee, 1871	17 mos ♀	Appeared to have abdominal pain; ascites	Hepatic veins closed off from vena cava by thin membrane, showing merely shallow dimples at these sites, many filled with tough thrombi; liver somewhat small, tough, with perihepatitis; signs of compensatory circulation		Primary cirrhosis of liver, extending to capsule, especially about hepatic veins
Schüppel, 1880	16 years ♀	Serofibrinous pleurisy, involving the right pleura (the only symptom mentioned)	Several hepatic veins of right lobe completely closed by organized thrombi; liver rather large; no perihepatitis spleen very large; no ascites; organized thrombi in longitudinal sinus and both transverse sinuses of dura mater		Possibly alteration of coagulability of blood, causing tendency to thrombosis

Hainski, 1884	22 years ♂	Ill for one year. Diagnosis of chronic peritonitis with pressure on vena cava and bile-ducts by new growth of fibrous tissue.	Organized thrombi in hepatic veins, no large vein emptying into vena cava; the walls of hepatic veins and vena cava thickened and surrounded by connective tissue which extended into liver; liver somewhat small, especially the right lobe; tough, congested, granular, and containing grayish-brown nodules; moderate perihepatitis; ascites, chronic enlargement of spleen, purulent peritonitis.	The nodules were found to be due to circumscribed hyperplasias, in the nature of regenerative growth of parenchyma.	Obliteration of the hepatic veins following thrombosis; the primary lesion was a chronic peritonitis which caused cirrhosis.
Lange, 1886	39 years ♂	Gradual illness nine mos. after pregnancy, ascites, abdominal discomfort, anasarca, large liver; ill five and a half months in all.	Hepatic veins in liver appearing as connective-tissue bands, many absolutely obliterated, some containing thrombi; one at a point of juncture of two veins held a calcareous nodule the size of a cherry; the inferior vena cava showing longitudinal thickening and numerous ostia of hepatic veins blocked; some branches of portal vein outside of liver thickened; liver normal size, congested, showed dark red and circumscribed yellowish areas; localized perihepatitis on upper surface posteriorly; ascites.	Hepatic veins showed thickening of intima, and in places also of adventitia, concentric in arrangement; somewhere stenosed, many obliterated; liver was congested, showing some increase of connective tissue in yellowish areas.	Primary chronic endophlebitis of hepatic veins, possibly syphilitic, although nothing in the history or physical data suggesting syphilis.
Kelynack, 1897	32 years ♂	For two years abdominal pain, later ascites, œdema of feet, icterus; four last pregnancies ending in abortion.	Only one ostium of hepatic veins into vena cava present, and that admitted only a small probe and had thickened edges; firm adhesions of liver posteriorly at site of vena cava and also to diaphragm; nutmeg liver, enlarged, tough; spleen large; ascites; left hydrosalpinx.	Hepatic veins contained thrombi, some recent, others partly organized; portal-vein branches were "full;" liver cells degenerated, and in part necrotic; also an increase of connective tissue.	The stenosis of the hepatic veins appears to have been caused by an inflammation of the capsule and of the tissue at posterior surface of liver; the veins becoming stenosed by the dragging of the adhesions; possibly the process was of syphilitic origin.
Chiari (1) v. Maschka, 1899	28 years ♂	Previously always well; twelve days previous to death abdominal pain, and for the last fourteen hours continuous vomiting so that poisoning was suspected; delivered of healthy child five months previous to death.	The hepatic veins from their ostia to the smallest branches were filled with red and gray thrombi; their walls in the neighborhood of the vena cava showed circumscribed thickening; they admitted only the finest probe; liver was very large, surface smooth; spleen large; ascites; scar on under surface of labia major.	The walls of the hepatic veins were thickened throughout, the proximal parts showing marked inflammatory changes and a proliferating endo-and periphlebitis, extending a short distance along the vena cava; near the ostia, the endophlebitis caused stenosis by projecting elevations of newly formed tissue; liver congested, no increase of connective tissue.	Primary chronic inflammation of the proximal ends of the hepatic veins, probably syphilitic in origin.

Case	Age and sex.	Clinical data.	Autopsy	Microscopic data.	Conclusions of the author
Chiari (2), 1899	55 years ♂	Woman had six children, five died in early youth, ill four weeks, ascites, œdema of legs, enlarged liver, sudden collapse.	The main trunks of hepatic veins entirely closed off by scar tissue from the vena cava, merely very small ostia being present; they contained thrombi, as did branches of portal vein, liver enlarged, capsule not thickened, spleen enlarged; hemorrhagic ascites and hemorrhagic infiltration of large intestine, partial aneurysm of left ventricle, obliteration of left coronary artery	The ostia of the main trunks of the hepatic veins showed a proliferating endophlebitis, the vena cava showed near the openings slight thickening of the intima and in places adherent thrombi, the portal veins contained thrombi; the liver showed congestion and slight increase of connective tissue	Primary obliterating inflammation of hepatic veins of long standing, the recent thrombosis causing the acute onset of symptoms, probably syphilitic in origin, for which the changes in the heart and coronary artery speak.
Chiari (3), 1899	29 years ♀	Abdominal pain, ascites, enlarged liver and spleen, death two months after onset, history of gonorrhœa	Hepatic veins stenosed by thickening of their walls so that merely a probe could be inserted; the obstruction was increased by thrombi at the ostia; the liver enlarged, capsule somewhat thickened, cut surface showing a larger dark red area corresponding to diseased veins and a smaller yellowish brown area wherein the veins were merely slightly dilated, the branches of portal vein were dilated but not thrombosed, spleen showed chronic enlargement, ascites; hyperæmia of stomach and intestines, hydrothorax	The hepatic veins in their proximal and peripheral parts showed proliferating endophlebitis with thrombus formation, causing stenosis or complete obliteration, the portal veins were slightly dilated, the liver tissue in the dark area showed atrophy due to stasis, with slight increase of connective tissue and extravasation of blood, in the lighter areas, stasis was not present	Primary endophlebitis of hepatic veins in the proximal and peripheral parts, secondary hepatic thrombosis with symptoms of stasis, possibly syphilitic in origin
Churton, 1899	26 years ♀	Two years' history of enlargement of abdomen, and later distention of the superficial abdominal veins not seriously ill until week before death, abdominal paracentesis showed blood tinged fluid, history of trauma on right side previous to enlargement of abdomen	The hepatic veins contained thrombi, one vein stenosed at entrance to vena cava contained an adherent thrombus, so that the ostium was difficult to find	Hepatic veins obstructed by thrombi united to vessel wall by connective tissue; cirrhosis of liver with formation of "new bile ducts"	Inclines to congenital syphilis as cause of stenosis of the hepatic veins and of cirrhosis of liver because no etiological factor could be found, and on the strength of successful mercurial treatment of a similar case.
Lazarus Barlow, 1899	13 years ♀	Four months previous to exitus noticed swelling of abdomen, later melæna and hæmatemesis, on examination, superficial abdominal veins found distended, liver enlarged,	An hepatic vein was occluded by a mass of cicatricial tissue on the upper surface of liver posteriorly, the ostia were normal, the tissue extended 1½ inches into liver parenchyma, trabeculæ running from it into liver the mass suggested a former gumma, numerous	Fibrosis of liver.	The hepatic veins obstructed by a cicatricial mass probably syphilitic in character, the thrombosis of the veins was secondary to the obstruction

		icterus present; tapped twice, sinking after second paracentesis.	branches of hepatic vein occluded by partially adherent thrombi; a localized suppurative peritonitis was present.		
Thran, 1899	32 years ♂	Two weeks previous to illness, gave birth to a child which died when three weeks old; complained of abdominal pain and enlargement; laparotomy for ovarian tumor revealed ascites and enlarged liver; later œdema of feet; four paracenteses performed.	Hepatic veins containing adherent thrombi; nothing said as to state of ostia: adherent thrombus 1½ cm. wide in inferior vena cava at diaphragmatic opening; liver small with greatly thickened capsule which sent broad connective-tissue bands into parenchyma; spleen much enlarged with thickened capsule; chronic parietal peritonitis.	Hepatic veins thickened, to a marked degree in parts, especially in inferior of liver: they also showed inflammatory infiltration, and almost all contained thrombi; from these veins connective-tissue strands could be traced to the liver capsule; these, however, not following the veins, but being irregularly distributed; liver extensively destroyed by growth of fibrous bands; portal veins not thrombosed.	The obstruction of the hepatic veins due to the pressure of the fibrous bands which resulted from the perihepatitis; the perihepatitis is thought secondary to a peritonitis probably gonorrhœal in origin.
Kretz, 1900	48 years ♂	Ascites for half-year previous to death; exploratory laparotomy revealed adhesions about gall-bladder and thickening of parietal peritoneum in bands; death three months later from marasmus.	Ostia of hepatic veins stenosed or obliterated by white endothelial masses, only four remaining intact; portal vein not affected; left lobe of liver small; right lobe large: the Spigelian lobe, projected almost like a tumor, was congested, and contained the normal hepatic veins running to vena cava.	Microscopic picture said to correspond to first of Chiari's published cases.	Thinks stenosis and obliteration of hepatic veins is due to exuberant scar tissue at the ostia of the hepatic veins, formed as result of trauma; frequently result of coughing; a second cause is diminished coagulability of blood in the hepatic veins; a third, the possible presence of infl'matory elements in the blood, as in syphilis or in infectious diseases.
Lichtenstern, 1900	20 years ♀	Since youth frequent attacks of diarrhœa and abdominal pain; a year previous œdema of feet; admitted twenty-three days previous to exitus, showing slight icterus, ascites, pain in epigastrium and loins; later œdema of feet and trunk, bloody urine passed, and toward end slight hæmoptysis.	The ostia of the two main hepatic veins and smaller upper veins obliterated, small ostia seen below, including those of veins going to Spigelian lobe; the obstructed veins contained adherent thrombi; portal veins normal; vena cava showed areas of thickening, and contained soft parietal thrombi; liver of normal size, capsule normal, contained red and yellow areas, the latter especially in region of portal trunk and in Spigelian lobe; ascites; spleen normal; hemorrhagic erosions of stomach; rectal hemorrhoids.	Hepatic veins obstructed by connective tissue at ostia; peripherally endo- and periphlebitis and organized thrombi; portal veins normal; vena cava showed signs of chronic inflammation; liver in red areas showed atrophy due to congestion and increase of connective tissue; in yellow areas cells were well preserved.	Hepatic veins closed by a primary inflammation of their walls leading to a seconday thrombosis; congenital syphilis probably the cause, although no other lesion of syphilis found.
Rendu et Poulin, 1901	35 years ♂	Abdominal pain and distention, ascites, dilated abdominal veins; death 3½ months after first symptoms; diagnosticat'd as cirrhosis of liver with thrombosis of vena cava.	Nothing said as to state of ostia of hepatic veins; vena cava and portal veins normal; liver somewhat enlarged, slight perihepatitis, marked cyanosis; great development of Spigelian lobe; spleen enlarged and indurated; thickening of peritoneum.	Marked congestion of liver with atrophy about the hepatic veins; hepatic veins showed obliterating endophlebitis and thrombosis.	Believes the phlebitis of hepatic veins due to alcoholism, but not sure whether it was primary or secondary to the thrombosis.

Case.	Age and sex.	Clinical data.	Autopsy.	Microscopic data.	Conclusions of the author.
Penkert, 1902	22 mos. ♀	Ascites, caput medusæ, enlarged liver and spleen; marked constipation.	Hepatic veins obliterated at ostia, slight retractions of wall of vena cava being present; the veins peripherally dilated and thrombosed; portal vein narrow, otherwise normal; liver enlarged, congested; capsule normal; anastomotic venous circulation.	Walls of hepatic vein thickened; liver congested, connective tissue increased, new formation and widening of biliary canals.	Obliteration of hepatic veins probably congenital; nothing pointing to syphilis.
Moore, 1902	25 years ♂	Ailing for three years; continuous pain in hepatic area for one month prior to admission; abdominal swelling noticed three days previous, also œdema of feet; dilated lower abdominal veins present; liver not palpable; no jaundice; eruption on legs *suspicious of syphilis*; paracentesis, followed by death five days later; ill five weeks in all.	Right and left hepatic veins only marked by shallow dimples and absolutely impervious for about one inch from ostia; they contained thrombi; liver small; no perihepatitis; it showed advanced central red atrophy; spleen small, congested; ascites; ductus venosus closed.	Hepatic veins closed by sparsely cellular connective tissue; media distinct, but showing intercellular fibrosis; intima thickened and obliterating the lumen; adventitia normal; hepatic veins thrombosed; liver showed central red atrophy, congestion, and hemorrhage.	Primary cause of obliteration of veins was endophlebitis, possibly due to syphilis; its localization to this area possibly due to the susceptibility of the veins in the vicinity of the ductus venosus to further obliteration.
Fabris, 1904	16 years ♀	Hemorrhagic ascites two months before death.	Large ostia of hepatic veins closed, only smallest existing; recent and older thrombi in hepatic veins; the vena cava somewhat whiter at site of obliterated openings; liver small, no perihepatitis; consistency increased; cut surface showed red zones and yellow zones. Caput medusæ.	No signs of proliferating endophlebitis of hepatic veins or of vena cava; walls normal; liver congested, with increase of bile-ducts.	Believes obliteration to be of congenital origin, as no reason to presuppose syphilis, and impossible to imagine an inflammation not leaving more evidence.
Meystre, 1904	28 years ♂	Three weeks previous to admission to the hospital complained of abdominal swelling; ascites found; operation, from which patient died two and a half months after onset of symptoms.	Hepatic veins contained thrombi; some were changed into connective-tissue bands, some showed thickened walls; the vena cava showed adherent thrombi overlying the obliterated ostia of the hepatic veins; some hepatic veins were patent; portal vein normal; liver congested and large; spleen enlarged; ascites.	Hepatic veins contained thrombi; some showed a thickened wall, the lining being irregular; in others the lumen was replaced by connective tissue; the vena cava inferior showed thickening of the intima at site of ostia of hepatic veins; portal veins normal; liver markedly congested, showed atrophy of parenchyma, combined with hypertrophy of some liver cells.	Primary endophlebitis of hepatic veins; no etiological factor present; author however inclines to syphilis as the cause.

An examination of the above columns shows that males and females have been affected in about equal numbers. Time of life brings with it no marked predisposition, as the ages of the patients have varied from seventeen months to fifty-five years, including the intervening decades; however, only three patients were over forty years of age. When we consider the clinical data, we find a general conformity in the course of the disease. The onset is generally gradual. Abdominal pain or discomfort, often most marked in the hepatic region, or an increase in the size of the abdomen, is almost invariably the first symptom, although for weeks or even months it may not be severe enough to cause the patient to seek medical advice. In some instances gastric symptoms have occurred later, but in a large majority these were not recorded. On examination the enlargement of the abdomen was found to be due to ascites, which rapidly increased and necessitated repeated paracenteses at short intervals. The fluid withdrawn had the characteristics of a transudate, but in 2 cases was slightly hemorrhagic. A caput medusæ, or distended abdominal vein, was often present. Œdema of the feet and anasarca occurred as late manifestations. Icterus was rarely noted, and then only of slight grade. The liver was invariably found enlarged, firm, and smooth in the early stage, but later small and slightly nodular. The spleen was large and firm. Temperature normal. Urine negative. The course of the disease rarely extended beyond six months from the date of onset. Such is the general clinical picture drawn from the 23 cases reviewed. The most marked variation from this type was the case of Lichtenstern, characterized by sudden onset and by gastric symptoms, resembling acute poisoning, followed by death within twelve days. When we review the conclusions, we find the greatest differences of opinion among the various authors. Most of them are in doubt, but each suggests a probable cause. Here, again, I believe, a table will most clearly show what these different opinions are:

	Perihepatitis.	Hepatitis.	Thrombosis.	Primary phlebitis.	Trauma.	Congenital anomaly
Frerichs	2					
Rosenblatt	...	1				
Gee	...	1				
Schüppel	...	...	1			
Hainski	1					
Lange	...	...	...	1		
Kelynack	1					
Chiari	...	...	...	3		
Churton	...	...	...	1		
Lazarus-Barlow	...	1				
Thran	1					
Kretz	...	...	...	...	1	
Lichtenstern	...	...	...	1		
Rendu and Poulain	...	...	...	1		
Penkert	...	...	...	...	...	1
Moore	...	...	...	1		
Fabris	...	...	...	...	...	1
Meystre	...	...	...	1		
	5	3	1	9	1	2

A glance at the above table will be sufficient to show how varied are the views as to the cause of the disease. I shall not discuss the question of etiology at this point, but shall proceed to the consideration of the case which came under my personal observation.

The patient was a girl, aged sixteen years, in the general hospital at Prague, on the service of Professor Pribram, to whom I am indebted for the careful history. Family history negative; no indications of syphilis. Had always been pale and weakly, and had never menstruated. Had had none of the diseases of childhood. Four years previously she had been a patient in another division of the hospital, suffering from marked abdominal distention, causing dyspnœa. She remained there for six months, during which time the ascites gradually disappeared under diuretic treatment and after frequent paracenteses. She was discharged, with the diagnosis of hypertrophic cirrhosis of the liver. From that time until October 1, 1904, had performed light housework at home. She then began to suffer abdominal pain, without apparent cause, and within three days experienced marked swelling of the abdomen and distention of the veins of the upper abdominal wall. Although she left perfectly well otherwise, these symptoms induced her to apply for admission to the hospital. The results of the physical examination may be summarized as follows:

Face pale, lips and extremities cyanotic. Clubbing of toes and fingers. The veins of the thoracic wall appeared like a meshwork, from the midst of which a markedly distended vein extended toward the abdomen. The heart showed great pulsation and dilatation to the right; sounds muffled, pulse soft and rhythmic. Lungs negative, abdomen distended, the maximum circumference being 87 cm. The vein extending upon the abdomen from the thorax contained tender nodules. Movable fluid, extending to within 2 cm. of the umbilicus, could be clearly distinguished. Liver was palpable 2 cm. below free border of ribs, consistency firm, surface somewhat nodular. Spleen extended 1 cm. below free border of ribs. Inguinal glands not enlarged. External genitals infantile. Lower extremities not œdematous.

October 29th. Circumference of abdomen 89 cm. Diarrhœa.

31*st.* Abdominal pain. Sleeplessness. Diarrhœa. Afternoon temperature yesterday, 100.8°. . Abdominal paracentesis with removal of 6 litres of slightly cloudy yellow fluid. The liver palpable three fingers below free border of ribs, hard, surface granular.

November 1*st.* Abdominal circumference 67 cm.

6*th.* Abdominal circumference 76 cm. Distention of veins includes upper abdomen, whereas yesterday it was confined to thorax.

7*th.* Abdominal circumference 78.8 cm.

8*th.* Abdominal circumference 79 cm. No œdema of feet.

9*th.* Severe abdominal pain.

13*th.* Death.

During her stay in the hospital the patient frequently had temperature as high as 100.8°. The urine was diminished in quantity and contained traces of albumin. The pulse tension (Gärtner) varied between 65 and 100. The treatment consisted in urea, theocin, and lecithin, at various periods, but all without effect.

Diagnosis. Cirrhosis hepatis and chronic peritonitis.

An autopsy was performed by Professor Chiari the day after exitus. The following is an abstract of the protocol: Body emaciated. Tonsils and cervical lymph nodes somewhat enlarged. Abdomen markedly distended; fluctuation present. The external genitalia were undeveloped and showed several superficial, firm, scars. From the mons veneris a bridge of skin, 3 cm. in length, stretched so as to form a furrow, extended to the clitoris. On opening the abdomen, greatly dilated subcutaneous and subperitoneal veins were apparent in the umbilical and in the epigastric areas. These ectatic veins were most numerous along the round ligament of the liver. Lungs adherent over entire surface; congested, slightly œdematous. Peribronchial nodes not enlarged. Pericardium contained 2 drachms of clear serum. Heart somewhat enlarged; the myocardium of light-brown color. Œsophagus dark red; its lower half showed markedly dilated submucous veins. The abdomen contained about 6 litres of clear serum. The peritoneum slightly thickened, smooth, glistening, especially thick over liver and spleen. Over the former it was "Zuckergussartig," or "iced," in patches. The omentum to a considerable extent adherent to the anterior abdominal wall in the epigastric area. It contained many dilated veins. Liver very small. Its width 20 cm., length 11 cm., thickness at point of greatest depth 7 cm.; its weight 950 gm. The surface showed numerous nodes, some 1 cm. in diameter. Consistency firm and tough. The cut surface of the liver showed everywhere numerous light-brown, circumscribed nodules, as large as a bean, projecting slightly from the surrounding parenchyma, which was grayish and of firmer consistency. No patent branches of the hepatic veins were apparent near the vena cava, but, instead, what seemed to be obliterated veins. The branches of the portal vein were patent throughout. The bile-ducts contained very dark bile. The trunk and main branches of the portal vein were dilated. The umbilical vein was present and patent for an ordinary anatomical sound. The intrahepatic portion of the vena cava was remarkable in that no ostia of the hepatic veins were visible on its inner surface. Ductus venosus arantii completely obliterated. Spleen weighed 170 gm., slightly enlarged. On section, tough, moderately vascular, trabeculæ soft. Malpighian corpuscles not evident to the naked eye. Kidneys moderately vascular, surface smooth, capsule non-adherent. In the perirenal tissue of the left side very numerous dilated veins, which could be traced into the mesentery

of the descending colon. Mucosa of urinary bladder somewhat œdematous. Accessory ovaries present on both sides. Stomach contained stringy mucus; mucous membrane pale.

The middle third of the small intestine was intensely red, its wall greatly swollen by hemorrhagic infiltration, and nearly 1 cm. in thickness. This part as well as the adjacent lumen contained much fluid blood. The veins of the mesentery of this section were dilated and contained obstructing thrombi, which were reddish and friable. The arteries were normal. The splenic vein contained fluid blood. The mucosa of large intestine was slightly reddened. Pancreas and adrenals were pale.

The pathologico-anatomical diagnosis was: Obliteration of the hepatic veins at the ostia in the inferior vena cava. Obstruction of the hepatic veins in the liver. Atrophy of the liver. Dilatation of the submucous veins in the lower part of the œsophagus, of the subperitoneal veins, of the subcutaneous veins at the umbilicus, with persistence of the umbilical vein. Adhesion of the omentum to the anterior abdominal wall in the epigastric region, and dilatation of its veins. Hydrops ascites. Thrombosis of the mesenteric veins with hemorrhagic infiltration of part of the small intestine. Accessory ovaries. Scars of external genitals. Abnormality of vulva.

Before detailing the result of the microscopic examination, I wish to describe more fully the macroscopic picture of the liver and the hepatic portion of the vena cava. The conformation of the liver and the appearance of its cut surfaces were striking. Two points were noticeable at a glance: the relative smallness of the left lobe and the relative greatness of the Spigelian lobe, which, viewed from the posterior surface of the liver, projected downward like a tumor mass from the body of the organ. This abnormal development of the Spigelian lobe must not be looked upon as an accidental condition, as several of the reported cases showed a similar local development. It is to be considered, in a measure, characteristic. Closer examination of the various cut surfaces of the liver showed that the light-brown or yellowish nodules mentioned in the protocol were distributed throughout the organ and that they varied in size from a pinhead to 2.5 cm.; that they were not more numerous in the large right than in the small left lobe; that they were round or oval in shape, and were almost invariably surrounded by a definite ring of firmer tissue, macroscopically suggesting connective tissue. We next followed and examined the hepatic veins, the inferior vena cava, and the branches of the portal veins as they appeared successively in the various horizontally cut segments of the liver. The obliterated hepatic veins could without difficulty be traced into the parenchyma, in some segments for 2 to 3 cm., extending from the vena cava as firm, gray bands, without lumen. The vena cava in the posterior surface of the liver showed four minute ostia, ranging

from a pinpoint to a pinhead in size. Three were at the upper angles of the vena cava, one to the left, two to the right, at the sites of the two main hepatic veins; the fourth was situated to the right of the median line at about the centre. The vena cava itself showed no stenosis. The portal vein likewise showed changes to the naked eye. Its right branch was obstructed by firm-contracting tissue not far from the porta hepatis; its left branch contained fresh thrombi, surrounded by reddish liver parenchyma.

The entire hepatic portion of the vena cava with the underlying liver tissue was removed for microscopic examination. Seven segments were thus obtained. Portions of the interior of the liver as well as of the spleen were also excised. All were hardened in alcohol and embedded in celloidin. Section after section was cut in order to follow an hepatic vein to its outlet, and to show the obliterated vessel in continuity with the vena cava. The Van Gieson and the hæmatoxylin and eosin stains were employed. However, a rapid examination of the unstained specimen often sufficed to show that the ostium of the vein had not been encountered. The obliteration was in all cases caused by connective tissue at the ostia, which was continuous with that of the intima of the vena cava. Up to this point the veins were to some extent patent. The obliterating connective tissue was dense, slightly cellular, and in parts contained lacunæ filled with blood. Careful search could not reveal any pigment therein, such as one should expect to find if the obliterating connective tissue had replaced a primary thrombus. The stenosis which existed in all cases beyond the point of obliteration was due to a proliferation of the intima. Here a thrombus was frequently found blocking the lumen. The media of the veins was well preserved, and ran in an uninterrupted course with that of the vena cava. Nowhere was a break therein to be seen as if trauma had caused a previous rupture of the fibres. The adventitia was also normal. The vena cava often showed a marked depression at the site of some obstructed ostia. Near these sites the connective tissue of its intima was thickened. At one point a partly organized thrombus was attached, but beneath it the connective tissue of the intima continued unbroken.

The liver parenchyma showed dilatation of the central veins, with atrophy of the liver cells in the centre of the lobules. There was a marked increase of connective tissue, which ran in bands through the parenchyma. Bile-ducts were numerous. The yellowish nodules, so striking in the macroscopic picture, proved to be composed of cells in a good state of preservation, showing no atrophy. They stained less deeply with eosin; but the nuclei took the hæmatoxylin well and were of average size. These areas were distinctive in showing no dilatation of the capillaries, a small rather than a large number of bile-ducts, and no increase of connective tissue. The spleen showed moderate congestion, but was otherwise normal.

From a clinical and a pathological standpoint this case is fairly typical. It must be regarded as one of long standing, having lasted at least four years. I consider that the patient was already suffering from an hepatic endophlebitis when in the hospital for the first time, although her disease was at the time diagnosed as hypertrophic cirrhosis. Later she improved, and the ascites remained away for the long period of four years, probably as the result of the compensatory circulation, which, as the autopsy showed, had established itself. The endophlebitis, however, continued, causing the stenosis of the ostia to become more and more marked, until finally obliteration occurred. This absolute closure of the hepatic veins, and barrier to the outflow of the large volume of venous blood from the liver, was the beginning of the end, and led to the sudden and persistent ascites which drained the strength of the patient.

The pathological findings serve to justify this interpretation. That we have to do with a case of long standing is borne out by the cirrhotic-like, granular liver, by the dense connective tissue obliterating its veins, and by the yellowish areas scattered throughout the parenchyma, which I look upon as regenerative areas, or so-called liver adenomata.

What cause can be assigned for this obliteration of the venous circulation, so strangely localized in one small area? No hereditary constitutional taint was disclosed; no previous illness of the patient existed upon which to cast the blame. Even the autopsy revealed little besides the venous obliteration and its immediate consequences upon the liver and the portal system. I believe that an obstruction of the hepatic veins can most certainly be caused by a perihepatitis, as happened in the cases reported by Frerichs[3] and others. But no such condition was found to explain this case. A primary thrombosis of the ostia I could not assume, partly because there was no reason for the formation of multiple thrombi in healthy veins, but still more because, as I mentioned above, no vestiges of a clot, no pigment, could be found at the site of obliteration. I have no doubt, however, that thrombosis also may be the primary cause of an obliteration through the formation of connective tissue. It has been urged by Kretz[20] that all these cases are due to trauma, to tearing of the hepatic veins, which are to be considered as constituting the insertion of the liver to the inferior vena cava; that small tears are brought about by coughing, and result in an exuberant scarring of the vessel wall, diminished coagulability of the blood in the hepatic veins, preventing thrombus formation. An argument of this nature cannot be discussed theoretically; everyone is entitled to his individual opinion as to the probable occurrence of such minute traumatic lesions. However, Kretz also bases his opinion upon the fact that microscopically he has seen a deflection and irregularity of the media of the obliterated veins at their insertion

into the vena cava. With this point in view I examined the muscular coat of the veins most carefully, but could nowhere find a break in the media. In every instance I could follow it unbroken and undeflected into that of the vena cava. For this reason I cannot accept a traumatic origin for my case and must look for another cause. The etiological table given above shows that two cases have been ascribed to a congenital closure of the hepatic veins. To grant this as the etiological moment we should have to conclude that such a lesion might exist without symptoms for twelve years, or, if we apply it to many of the other published cases, well on into middle life. But what I believe furnishes absolute evidence against this origin is that the ductus venosus arantii was not patent. As Lazarus-Barlow[12] well says, this would not become obliterated under such abnormal conditions. Moore[19] and Rolleston[23] modify the congenital point of view by suggesting "a spread of the process of obliteration of the ductus venosus to the hepatic vein orifices—an excess of the obliterating process." This might well be true for the obliteration of the ostium of the left main hepatic vein, or for the vena cava at this point, but a study of the anatomical relations of this region does not allow me to believe that all the large hepatic ostia could be obliterated without causing some stenosis of the vena cava itself.

All the cases which I have considered above, as well as the one just reported have been uncomplicated by any narrowing of the hepatic vena cava. I now come to the last cause to be considered: a chronic endophlebitis. Clinically, my case and the majority of the cases, were slowly progressive in character. Pathologically, such change was found as is generally ascribed to chronic inflammation; a proliferation of dense connective tissue such as blocked the obliterated veins. Of all the causes advanced this one seems the most probable. We cannot, however, pronounce this view absolutely; as we are unable to define the exciting inflammatory cause. All but one of the writers who believe an inflammation to be the basis of the disease ascribe the changes to syphilis. However, to whichever opinion we incline, we must not lose sight of the fact that a close study of the cases shows that, with the exception of Chiari's first and, possibly, second case, none showed any other definite syphilitic lesions. If syphilis be regarded as one cause of the inflammation, it is probable that it is not the only one, but that inflammatory toxins of other natures can produce a like effect upon the lining of the hepatic veins.

As regards the diagnosis, I believe that I can venture the statement that no case of this disease has been correctly diagnosed. The cases have generally been confused with cirrhosis of the liver, although the ascites has also led to the diagnosis of tuberculosis or carcinoma of the peritoneum, and even to that of ovarian cyst. The two fulminating cases, characterized by vomiting, prostration,

and a rapid course, were diagnosed as due to poisoning in the one instance and to intestinal obstruction in the other. It would lead to nothing to consider these cases further, as, in the present state of our knowledge, we could not hope to suspect the underlying condition when presenting itself in this violent form. But a study of the other cases shows that although this disease cannot be absolutely diagnosed, it can and should be thought of when a certain group of symptoms presents itself.

We have the typical signs of cirrhosis of the liver: ascites with dilated abdominal veins, sometimes a caput medusæ, very slight or no icterus, a palpable liver in the early stages, and a palpable spleen. What can lead us to suspect an obliteration of the hepatic veins as the cause? In the first place the history of the patient; the absence of all etiological cause for a cirrhosis. This is important. If, in addition to this, the patient is a very young individual, as is often the case, our suspicion should be awakened to the possibility that we are not dealing with an ordinary cirrhosis, a disease of late middle life.

Furthermore, an important differential diagnostic point is the occurrence of pain over the hepatic area, or localized in the upper abdomen, which is one of the most constant and earliest symptoms of endophlebitis, but exceptional in cirrhosis. Again, as Quincke[14] says, the rapidity with which the swelling of the liver and ascites develop is of importance. The patient with chronic endophlebitis will generally state that the swelling of the abdomen has showed itself within a few weeks or even a few days. In striking contrast to this is the gradual onset of abdominal swelling and ascites in cirrhosis. Later in the course of the disease the rapidity with which the ascitic fluid collects, the frequent necessity of paracentesis, point to an essentially obstructive lesion. All these symptoms, however, although brought about by a disease of the hepatic veins, are directly dependent upon the secondary obstruction in the portal system and are typical of portal obstruction. We cannot distinguish between them, and it is only the lack of cause for a portal obstruction that should make us consider whether this condition is not the result of a primary hepatic obliteration.

Beyond this we cannot go in the differential diagnosis. The paucity of cases presented for review does not allow of greater generalization. I am certain, however, that if this disease is taken into consideration at the bedside, and looked for in the postmortem room, we shall before long find many more upon which to base an opinion. Again, I wish to lay stress on the importance of opening the hepatic portion of the vena cava, and examining the ostia of the hepatic veins in all cases, but especially in those where ascites has been present.

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A CASE OF EXCISION OF THE HEAD OF THE HUMERUS FOR CONGENITAL SUBACROMIAL DISLOCATION OF THE HUMERUS.[1]

By John B. Roberts, M.D.,

of Philadelphia.

A boy, aged three years, was operated upon on December 5, 1903. The head of the bone could be seen and felt under the acromial process, and the humerus was firmly abducted from the chest with the external condyle pointing almost directly forward instead of

[1] Read by title at the meeting of the American Surgical Association, July 5, 1905.

outward. The position of the arm and attached forearm was due to the rotation of the humerus, caused by the displacement of the head of the bone. Voluntary elevation of the arm was impossible and other movements were restricted. It was, therefore, deemed advisable to attempt replacement of the head of the bone by opera-

FIG. 1 FIG. 2



Congenital subacromial dislocation of the left humerus. Boy, aged three years. Observe abduction and inward rotation of humerus. Before operation. *a*. Olecranon points directly outward and external condyle forward. *b*. Head of humerus.

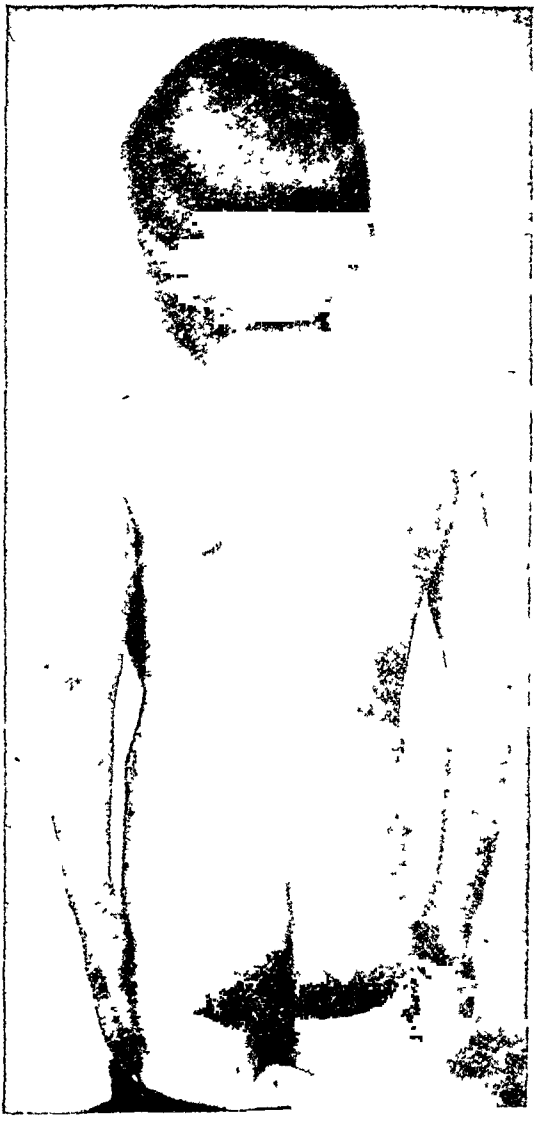
tion, and in case of failure, to excise the humeral head. A vertical incision, three inches long, was made over the prominence below the acromion, through the fibres of the deltoid. The round head of the bone was covered by a deeper muscle, which was probably the infraspinate. After opening the capsule and exposing the head, attempts were made to reduce the dislocation. As reduction was

impossible, an incision about three inches long was made in a vertical direction through the deltoid on the front of the shoulder and carried down to the deep layers of muscle. Counterpressure upon the bone was made here in the endeavor to aid in the reduction of

FIG. 3



FIG. 4

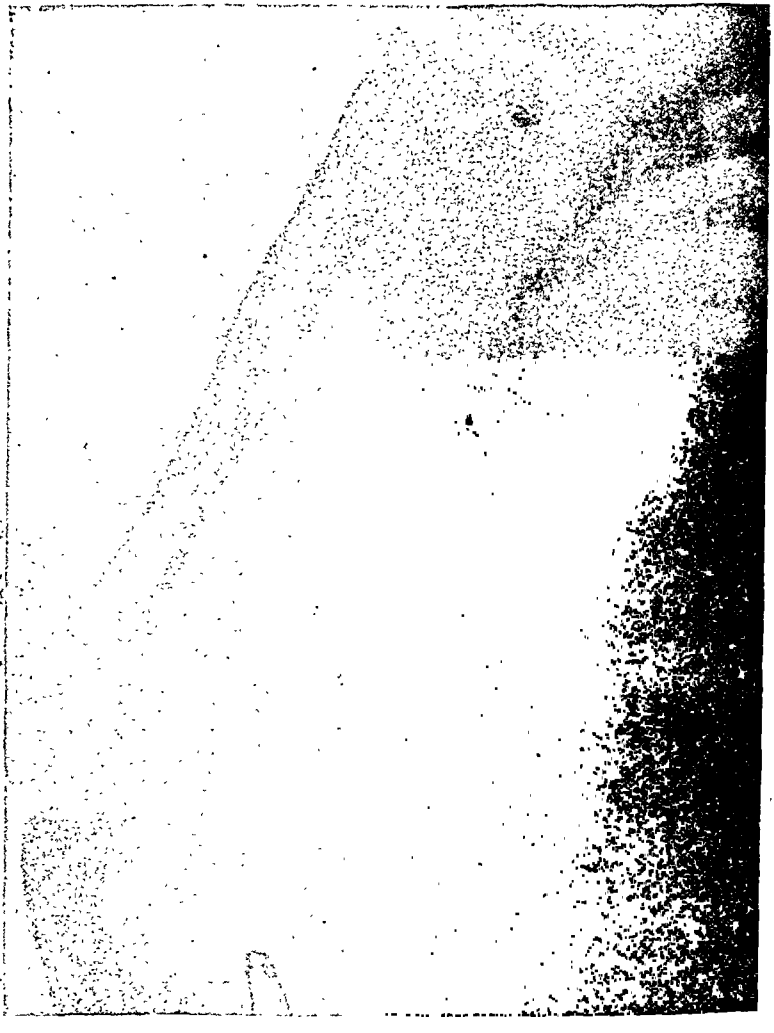


Congenital subacromial dislocation of left humerus Same patient as Fig. 1. Photograph taken about eighteen months after excision of head of the humerus Observe improved position of elbow, but the shortening of humerus. *a*. Body of biceps unnaturally prominent because origin of one head is lost

the luxation. All these efforts were unavailing. The glenoid cavity itself was not exposed, because of the depth of the wound and the youth of the child. The condition seemed to make it evident that only a prolonged and bloody dissection would thoroughly expose

the glenoid cavity. I therefore came to the conclusion that excision of the head of the bone would give probably as good, if not a better, joint with less risk. Reduction with stiffness of the joint seemed to me to be but little better than the present condition. The head of

FIG. 5

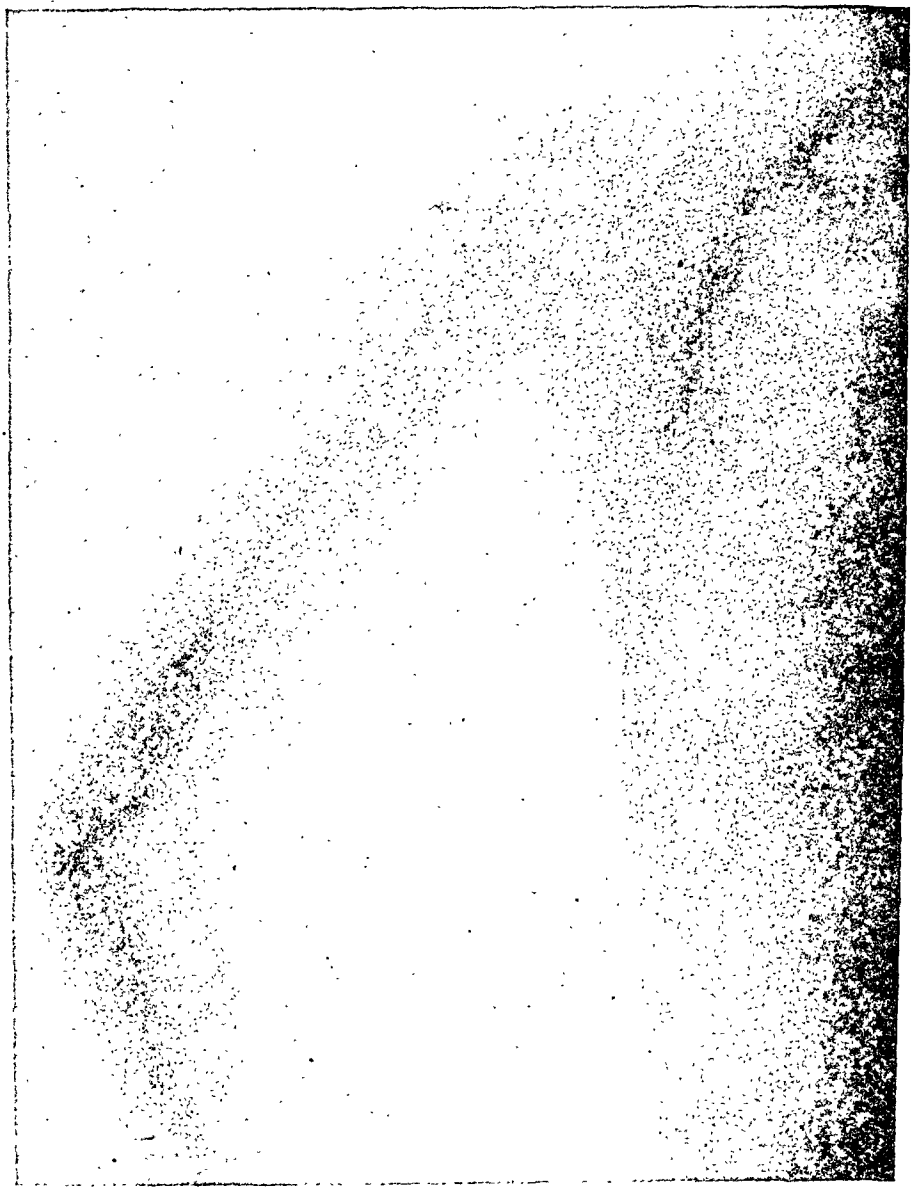


Congenital subacromial dislocation of the left humerus. Same patient as Fig. 1. Observe abduction rotation of humerus, shown by the elbow being held away from the thorax, with olecranon pointing outward.

the bone was thoroughly exposed and excised through the posterior wound. The section was made with a small saw close to the head, so as to remove as little as possible of the shaft. In removing the excised fragment the long head of the biceps was divided in the

depth of the wound close to the axilla. The rotation of the bone caused the bicipital groove and its enclosed tendon to be almost directly opposite the incision made on the back of the joint. The

FIG. 6



Congenital subacromial dislocation of the left humerus. Same patient as Fig. 1. Skiagraph taken about three months after excision of the head of the humerus. Observe the absence of abnormal rotation of the humerus, which is seen in the skiagraph taken before operation. The abduction of the humerus seen here is voluntary.

division of this muscle was not recognized at the time. Both cuts were closed without drainage and the forearm supported by a sling. Union occurred by first intention.

When seen on March 2, 1904, the motion of the arm was good, though the limb was weak. The child had a tendency to aid it by lifting it with the right hand when told to raise it. He could touch the ear on both sides and the back of the head with the left hand if the arm is aided a little with his own right hand or an attendant's hand. This aid is needed especially when he touches the right ear. His mother said that he always helped the dislocated arm with the other hand, and that this habit was still kept up, though there was much less need of such assistance. Frictions and calisthenic exercises were recommended.

When examined June 1, 1905, the notes made were as follows: The left humerus is considerably shorter than the right. Passive motion elicits grating at the shoulder-joint. There is possible moderate rotation, though it is not as complete as on the right side. Elevation of the humerus is restricted, and the scapula moves with the humerus, if passive movements are at all extensive. There is still some rotation forward of the humerus when the arm is held at rest, so that the external condyle presents rather more forward than in the normal arm. The boy can now touch the back of his ear and neck on the left side. He can put his hand to his mouth, which he could not do before, and he can clasp his hands behind his back in the lumbar region. The normal arm from the acromion to the external condyle is six and one-half inches long. The left arm from the acromion to external condyle measures five and three-quarters inches. The belly of the biceps stands out prominently in the middle of the arm. This is due largely to the division of its long head at the operation. The forearm is somewhat atrophic, and the left hand seems slightly smaller.

He cannot lift the left hand over his head, but he can nearly touch the occipital region. He uses the right hand to lift the left arm when he desires to put the left hand on top of his head.

His general health is good and the scapular muscles seem about as well developed as on the other side. The deltoid seems to be a little more prominent than on the other side.

It is doubtful whether he can voluntarily bring the left elbow in contact with his ribs, but he can put the left hand on the right shoulder and bring the elbow and humerus close to the ribs. It is difficult to determine the exact amount of adduction, because of the youthfulness of the child and the difficulty of making him understand.

The deformity of which this case is an illustration is unusual. There has been considerable discussion as to how many of such cases are traumatic dislocations caused by injuries at the time of delivery. I see no reason to doubt that a large proportion of them are the result of intrauterine conditions. Accidental subspinous or subacromial luxations are so rare in both adults and children that it seems to me reasonable to look upon them, when found immediately

after birth, as true congenital conditions. It is true that violence during delivery may cause the lesion, and also that such displacements may be the result of a muscular paralysis occurring before birth or during delivery. It is quite possible, therefore, that some of the cases reported belong to these groups. The occurrence of this posterior displacement occasionally in both shoulders of the same infant and at times in more than one child of the same family is strong evidence, in my opinion, of the prenatal origin of the bony abnormality. I have not been able to find many cases recorded in the literature which bear rigid analysis. Stimson[1] says that he has seen 4 or possibly 5 cases. Phelps had seen, according to Stimson, 6 or 7. Scudder[2] reports 2; Küster,[3] 1 bilateral case; Gaillard,[4] 1; R. W. Smith,[5] 1 bilateral case; Cumston,[6] 1; Peckham,[7] 2; and I reported a previous case ten years ago to this Association.

VOLVULUS OF THE CÆCUM AND ASCENDING COLON.

By Alfred C. Wood, M.D.,
of Philadelphia,
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Mrs. O. B., aged thirty-four years, widow (Philadelphia Hospital, No. 1756), was admitted from the out-wards on account of severe abdominal pain and vomiting, September 14, 1903.

Her statement was as follows: On the evening of September 13, 1903, she lifted a heavy woman into bed, and afterward "felt badly," although she did not report the matter and did not do anything to get relief. About 11 o'clock on the following morning she felt a sudden, very sharp, lancinating pain in the abdomen. The pain continued, was paroxysmal in character, and was so severe as to cause her to cry out; it seemed distributed equally over the abdomen. Vomiting began soon after the pain, and has since been almost continuous. The bowels acted regularly and normally until the

[1] Fractures and Dislocations, ed. 1905, p. 606.

[2] Archives of Pediatrics, 1890, vii. p. 260.

[3] Ein Chirurgisches Triennium, 1882, p. 256; also in Berlin. klin. Woch., January 6, 1879, Bd. i. p. 9.

[4] Mém. de l'acad. de méd., 1841, p. 703; quoted fully by Alph. Robert in Des vices congénitaux de conformation des articulations, Paris, 1851, p. 53.

[5] An Essay upon the Original or Congenital Luxations of the Upper Extremity of the Humerus, Dublin Medical Journal, 1839, vol. xv. p. 249; also mentions the case in his Treatise on Fractures, Dublin, 1854, p. 256.

[6] The American Journal of the Medical Sciences, June, 1903.

[7] American Journal of Orthopedic Surgery, April, 1905, p. 386.

beginning of the present trouble, two days ago, since which time there has been no movement. The menstrual function is normal, the last period having been two weeks prior to this attack. The patient has one child living and in good health. There have been no miscarriages. There is no venereal history.

Physical Examination. The patient looks older than the age she gives. She is well developed and fairly well nourished, the face is pale, and the expression denotes acute suffering. The tongue is moist and clean. The pupils are small, equal, and contract normally. The lungs are normal. The heart sounds are regular, but weak; pulse 86 and compressible. The abdomen is much distended, apparently more on the left side than the right. The surface above the umbilicus is red from the effects of a mustard plaster; striæ gravidarum are prominent. The abdomen is everywhere board-like to the touch, no definite mass can be felt, and palpation gives the patient severe pain. The abdominal pain is reflected up to the region of the right shoulder. Flexing the thighs upon the abdomen gives marked relief. During the severe pains the patient groans and tosses about the bed.

Nausea and vomiting are almost constant. The vomitus is black, watery, and has a sour odor, but is not bilious or fecal. There is a great deal of retching. The skin is bathed in a cold perspiration. The leukocyte count is 26,400. The urine contains albumin and hyaline and granular casts.

Operation, September 15th. The writer did not have an opportunity of examining the patient until she had been placed under the influence of ether on the operating table. It was then possible to palpate a large, rounded, uniform mass, occupying chiefly the right lower quadrant of the abdomen. The abdomen was opened through an incision along the outer border of the right rectus muscle. A quantity of dark-colored, offensive fluid escaped from the abdominal cavity when the peritoneum was opened. Peritonitis had evidently already begun. The tumor was found to be a distended loop of intestine, which on inspection proved to be a volvulus. The bowel forming the volvulus was lustreless and of a deep purple color, looking almost gangrenous. There did not seem to be anything but gas in the distended loop. The bowel was delivered from the abdomen and untwisted without incising, the distention disappearing as reduction took place. The volvulus was composed of the cæcum and part of the ascending colon. Every portion of both of these structures was so devitalized that resection was imperative. The ileum adjacent to the cæcum appeared to be but little affected, there were no gross changes that would lead one to question its vitality. The latter was therefore divided near the ileocæcal valve and the colon at the hepatic flexure, the two ends being united without difficulty by means of Murphy's large-size button. The gauze pads which had surrounded the field of operation were

removed, the abdominal cavity was thoroughly flushed with sterile water, and the wound closed by interrupted sutures.

The operation was concluded about 5 P.M. At 7 o'clock the pulse was recorded as 84, temperature $99\frac{2}{5}°$, respiration 28. There was but little change until 9 o'clock, when the pulse and respirations rose rather rapidly, and the temperature gradually. From this time on the patient failed steadily, and died at 4 o'clock the following afternoon, twenty-three hours after operation, the pulse having reached 180, the respiration 56, and the temperature $103\frac{3}{5}°$.

The autopsy showed that several inches of the ileum adjacent to the point where it had been divided had become gangrenous, and there was a general peritonitis.

COMMENT. A careful study of the relations of the parts affected was impossible, on account of the necessity of concluding the operation as speedily as possible. The conditions found, however, appeared to be as follows: The cæcum was at the upper extremity of the tumor and internal to the colon. A single turn relieved the volvulus.

The mechanism of this case seems to have been as follows: first, the descent of a loop of ascending colon, made possible by an abnormally long mesocolon; the falling of the colon revolved the cæcum on its anteroposterior axis, so that it first pointed toward the right and, as the process continued, upward. As distention in the loop took place the cæcum was forced upward along the parietal wall, carrying the ileum with it, until it finally crossed over the ascending colon anteriorly.

Treves divides volvulus of the ascending colon and cæcum into three categories:

"1. A twist of the ascending colon around its own axis.

"2. Twists brought about by an abnormal loop formed by the ascending colon and cæcum with a long and distinct mesocolon.

"3. Twists of the cæcum 'upon itself' or about its own axis."

The case here reported belongs to the second division. According to Treves, more than two-thirds of all cases of volvulus of the intestine occur as a result of rotation of the sigmoid flexure on its mesenteric axis. The above case is of interest on account of its rarity. Treves states that twists of the cæcum and ascending colon are among the least common varieties of intestinal obstruction.

As to the causes of volvulus of the sigmoid, the same authority states that chronic constipation ranks first; while in the second and third of the varieties just mentioned some congenital abnormality has been present in all the reported cases, without which the twist could not have occurred. This abnormality related either to defective development of the cæcum—that is, to a faulty position—or to an unusually long mesentery.

Peritonitis is a very constant and early complication in these cases. When the loop of intestine is greatly distended the overlying peritoneum may be torn in one or more places. Such a rent is to be observed in the accompanying illustration:



Increased interest has been given to this subject by Corner and Sargent,[1] who publish an anaylsis of fifty-seven cases. They

[1] Annals of Surgery, January, 1905

emphasize the point that while acute examples of this condition have been recognized for a long time, it does not seem to have been observed that subacute and chronic forms also occur. In illustration, the duration of the symptoms in the fifty-seven cases was as follows:

Less than 1 day	7 cases.
1 to 2 days	2 "
2 " 3 "	6 "
3 " 4 "	6 "
4 " 5 "	4 "
5 " 6 "	4 "
6 " 7 "	1 "
8 " 14 "	10 "
15 " 21 "	3 "
Unstated	10 "
Described as "chronic"	4 "

Moreover, the authors state a number of the patients had suffered premonitory attacks of abdominal pain and vomiting, the last attack differing only in degree from the previous ones.

Examination of the records shows that a considerable number gives accounts of previous and recurrent abdominal trouble, and even of definite intestinal obstruction, which have passed off. Such attacks vary in duration from a few minutes to several days. Some patients have been subject to them for months or years; while in other cases the premonitory attacks have preceded the final acute one by only a few weeks or days.

The most constant symptom is abdominal pain, which naturally varies greatly in character and intensity in different cases. The most frequent concomitants are vomiting and inaction of the bowels, as regards both flatus and feces. The abdomen may rapidly become distended; in some instances a resonant tumor may be recognized. As regards age, the cases were distributed as follows:

Under 5 years	3 cases.
5 to 10 "	1 "
10 " 20 "	7 "
20 " 30 "	15 "
30 " 40 "	14 "
40 " 50 "	7 "
50 " 60 "	4 "
60 " 70 "	2 "
70 " 80 "	1 "
Adult	1 "
Unstated	2 "

The extremes were 19 days and 80 years.

FURTHER OBSERVATIONS ON THE RELATION OF LESIONS OF THE GASSERIAN AND POSTERIOR ROOT GANGLIA TO HERPES OCCURRING IN PNEUMONIA AND CEREBROSPINAL MENINGITIS.[1]

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(From the Pathological Laboratory of the Lakeside Hospital, Cleveland, Ohio.)

Introduction. At the meeting of the Association in 1902 I reported two cases of herpes, one of the lip and nose and the other of the trunk, occurring in acute croupous pneumonia, in both of which the sensory ganglia supplying the areas affected with herpes showed acute degenerative and exudative changes.

At this time I suggested that careful examination would disclose similar lesions in the sensory ganglia supplying areas affected with herpes, complicating other infectious diseases, especially cerebrospinal meningitis, typhoid fever, and malaria.

Since then I have had opportunity to examine at autopsy two cases of cerebrospinal meningitis and one case of pneumonia with herpes. In all three cases definite lesions were found in the sensory ganglia supplying the herpetic areas.

In addition, I have seen in the wards of the Lakeside Hospital, in the service of my colleague, Dr. John H. Lowman, two unusual examples of facial herpes complicating non-fatal pneumonia.

Report of Cases: Herpes Complicating Pneumonia.

Case I. *Summary: Herpes of the skin of the right side of the nose, especially marked near the inner canthus of the right eye, occurring in a child with pneumonia complicating typhoid fever; degenerative and exudative changes in that portion of the right Gasserian ganglion, giving rise to the nerves supplying the skin of the upper portion of the nose; left Gasserian ganglion normal.*— T. W., male, aged four years, was admitted on March 26, 1903, to the Lakeside Hospital, service of Dr. John H. Lowman, after an illness of thirteen days. He was drowsy and apathetic and breathed rapidly. Physical examination showed signs of consolidation of the upper lobe of the right lung. The spleen was enlarged. Widal reaction positive. There was a well-marked crop of herpes near the inner canthus of the right eye. On the morning of March 31st

[1] Read by title at the Twentieth Annual Meeting of the Association of American Physicians, Washington, D. C., May 17, 1905.

sloughing of the right cornea was recorded. Death occurred at 11.45 P.M., March 31, 1903.

Clinical Diagnosis. Typhoid fever, acute croupous pneumonia of the right lung, herpes of the right side of the nose, sloughing of the right cornea.

Anatomical Diagnosis. Typhoid fever (ileotyphus), hyperplasia of the mesenteric lymph nodes and the spleen, acute croupous pneumonia of the right lung (all three lobes), acute serofibrinous pleurisy, emphysema of the left lung, focal necrosis of the liver, cloudy swelling of the heart, liver, and kidneys. Herpes of the right side of the nose, sloughing of the right cornea, congestion, hemorrhage and acute degenerative and interstitial inflammation of the right Gasserian ganglion.

Bacillus typhosus was isolated from the spleen and gall-bladder, and staphylococcus pyogenes aureus from the lungs, heart blood, and liver.

The autopsy was begun ten hours after death. The detailed description of the autopsy findings will be omitted. The Gasserian ganglia were of ordinary size. The veins about both were congested. The capsules and nerves of both were free from signs of inflammatory exudation. There was no meningitis.

Histological Examination of the Gasserian Ganglion and the Fifth Nerve. Right Gasserian Ganglion: There was marked congestion of the veins along the convex border of the ganglion. Here, corresponding to a portion of the origin of the superior maxillary muscle of the fifth nerve, there was an area showing marked degeneration of the ganglion cells and infiltration of the interstitial tissue with leukocytes. The fifth nerve and its branches showed no degenerative or inflammatory changes. The *left Gasserian ganglion* and fifth nerve were normal.

CASE II. *Summary: Herpes of the right upper and lower lips, the right upper and lower eyelids, and the right side of the neck*—i. e., *of the regions supplied by the right Gasserian and the right third cervical ganglia, occurring during an attack of acute croupous pneumonia, ending in recovery.*—Mrs. H. C., aged fifty-eight years, was admitted to the Lakeside Hospital, service of Dr. J. H. Lowman, on May 7, 1904, with the physical signs of acute croupous pneumonia of the lower lobe of the right lung, which later involved the entire lung. She was taken ill on May 3d, with a chill and pain in the right side of the chest. On admission there was an extensive crop of herpes involving both the upper and lower lips (most marked on the right side) and the right upper and lower lids of the right eye. There was conjunctivitis with granular lids on this side.

On the sixth day of her illness a crop of herpes appeared on the right side of the neck two inches below the angle of the jaw.

The patient recovered, so there was no opportunity to examine the sensory ganglia. The herpes ran the usual course of the so-called

herpes zoster. In the light of our previous cases and of those of meningitis reported below, it is evident that the patient had extensive lesions of the right Gasserian and of the right third cervical posterior root ganglia.

CASE III. *Summary: Herpes of the left lower eyelid occurring in the course of acute croupous pneumonia.*—In this man, also in the service of Dr. J. H. Lowman, there was herpes orbicularis occurring in a typical attack of acute croupous pneumonia, ending in recovery.

Herpes Complicating Cerebrospinal Meningitis.

CASE IV. *Summary: Epidemic cerebrospinal meningitis, with herpes of the right side of the face and neck, and of the left upper lip, and the vestibule of the nose and of the head and soft palate, regions supplied by both Gasserian and the third right cervical posterior root ganglia.*—D. L., male, aged twenty years, entered the Lakeside Hospital, service of Dr. H. H. Powell, October 27, 1904, complaining of severe pain in the head and the right side of the body. He had always been well until October 20th, when he was taken ill with chilly sensations and pain in the head and body.

Four days later he suffered severe burning pain in the right side of the neck. Later at this point a rash developed, followed by a large crop of herpetic vesicles. On admission there was marked rigidity of the neck, hyperæsthesia, and marked herpes, involving a large area on the right side of the neck below the angle of the jaw. Herpes later developed on the right cheek, near the ear. Kernig's sign was present. The diplococcus intracellularis meningitidis was isolated from fluid removed at lumbar puncture. Death occurred November 11, 1904.

Clinical Diagnosis. Epidemic cerebrospinal meningitis with facial and cervical herpes.

Autopsy. The autopsy was begun one hour after death.

The following is abstracted from the autopsy protocol: The body is that of a white male, 167 cm. long. There are extensive herpetic eruptions involving the right cheek just in front of the ear, the right side of the neck, the left upper lip and vestibule of the left nostril and the mucous membrane of the hard and soft palate on both sides. The eruption of the lip and nose is in the vesicular stage. That on the face and neck is covered with dry crusts. In the mouth the eruption is macular and pustular.

Nervous System. The dura and pia arachnoid are congested. Over the surfaces of the medulla and pons there is a thick fibrinopurulent exudate of gelatinous consistency. At the base of the brain and in the lateral ventricles there is a considerable amount of turbid fluid. The nerves at the base of the brain are enveloped in the exudate.

Gasserian Ganglia. Both are surrounded by a thick purulent exudate. The bloodvessels are congested.

Spinal Cord. The membranes of the cord are bathed in a turbid fluid. The pia arachnoid is covered with a fibrinopurulent exudate, which extends to many of the nerve roots. The cord on section shows no appreciable changes. The third right cervical ganglion is larger than its fellows of the opposite side.

The other organs show no changes of present interest and their description is, therefore, omitted. The diplococcus intracellularis meningitidis was isolated in pure culture from the cerebrospinal exudate.

Microscopic Examination of Nervous System. The central part of the convex border of the ganglion—*i. e.*, that portion corresponding to the origin of the superior maxillary branch and possibly a portion of the inferior maxillary branch is the seat of extensive degeneration and destruction of ganglion cells, with leukocytic infiltration.

The affected ganglion cells are cloudy, stain poorly and are often hyaline. Some cells are swollen and others are shrunken. In those less affected the nuclei show no changes. In markedly degenerated cells the nuclei may be eccentric and some stain diffusely. The leukocytic infiltration is most marked in the interstitial tissue, but some ganglion cell spaces contain leukocytes. The plexus of nerve fibres coming from this portion of the ganglion are surrounded with a fibrinopurulent exudate. Sections of the sensory root of the fifth nerve show no recognizable changes, when stained in either methylene blue and eosin or by Weigert's myelin sheath method.

Left Gasserian Ganglion and Fifth Nerve. In this ganglion similar changes are present and in the same portion of the ganglion as described in the right. The sensory root of the fifth nerve shows small scattered areas of cellular infiltration.

The Third Right and Left Cervical Ganglion and Posterior Root Nerves. The right ganglion is the seat of numerous small areas of ganglion cell degeneration and of interstitial and periganglion cell cellular infiltration. The changes are best marked at the distal end and at one side of the ganglion. The right posterior root is surrounded and infiltrated by large numbers of leukocytes. The left posterior root is similarly but less intensely affected. The left ganglion is normal.

Sections of the cord made at various levels show marked meningitis. The other ganglia are free from changes.

Anatomical Diagnosis. Acute epidemic cerebrospinal meningitis, due to diplococcus intracellularis meningitidis. Degenerative and exudative changes in both Gasserian and in the third right Gasserian ganglia, herpes of the hard and soft palate, the left upper lip, the vestibule of the nose, the right cheek and the right side of the neck.

In regard to the lesions in the third right cervical ganglion, it is clear that the infection made its way along the posterior nerve roots. In the case of the Gasserian ganglia the route of the infection is not

so clear, but it is evident that the inflammatory process is more marked in the nerve bundles about the ganglion than in the latter structure itself. It is to be noted that the distribution of the herpetic lesions corresponds exactly with the ganglionic lesions.

CASE V. *Summary: Trifacial neuralgia of the right side, suppuration of the sphenoidal sinuses; brain abscess and acute cerebrospinal meningitis, due to an actinomyces; herpes of the posterior and lateral cervical regions of the side.*—E. W., coachman, aged forty-six years, was admitted on February 11, 1905, to the Lakeside Hospital, service of Dr. J. H. Lowman, complaining of severe trifacial neuralgia of the right side. He denied having had any previous illness, except whooping-cough in childhood. He stated that he had been in good health until two weeks before admission, when his illness began with severe headache and backache. There was a slight chill, but no cough or other symptoms of disease of the respiratory tract. Ten days before admission he developed some pain, paroxysmal in character, and tenderness on pressure of the right side of the face and head. Remedies applied in the hospital relieved this pain.

On February 13th, two days after admission, he developed symptoms pointing to brain abscess on the right side and to cerebrospinal meningitis. At lumbar puncture 70 c.c. of turbid fluid were obtained. From the fluid Dr. Louis W. Ladd isolated an actinomyces, which will be described in a future paper.

At this time there appeared well-marked herpes of the skin over the capital portion of the trapezius muscle and the angle of the scapula on the left side. There was no herpes elsewhere. Death occurred February 17, 1905.

Clinical Diagnosis. Abscess of the right side of the brain, cerebrospinal meningitis due to an actinomyces. Herpes of the neck.

Autopsy. The autopsy was begun a few hours after death. As this case will be reported in full in another paper, the present descriptions of the pathological findings will be restricted to the lesions of the right fifth nerve and Gasserian ganglion and the third and fourth cervical ganglia and their roots.

Abstract of the Autopsy Protocol. The right Gasserian ganglion is of ordinary size. The sensory nerve of the fifth nerve is swollen and is marked by dark-red streaks. The veins about the ganglia were markedly dilated.

Histological Examination. Some of the veins of the capsule of the ganglion are found to be congested, but there is no hemorrhage. The ganglion cells stain normally, are not shrunken or invaded by cells. There is no infiltration of the interstitial tissue. In sections of the root of the fifth nerve stained with eosin and methylene blue, marked inflammatory lesions are found. The small veins and capillaries are congested and widely dilated. There are numerous areas of hemorrhage, which distort the nerve tissue. In many

sections there are large accumulations of red blood corpuscles, leukocytes, and large oval cells about the dilated bloodvessels. The large cells, which are most numerous are evidently proliferated connective-tissue cells. In sections stained by Weigert's myelin sheath method, there is marked breaking up and even disappearance of myelin sheaths about many of the fibres about the areas of hemorrhage and inflammation. The left Gasserian ganglion and fifth nerve show no pathological changes.

Third Cervical Ganglia and Posterior Roots. The left ganglion is larger than its fellow of the opposite side. Histologically, the changes are marked. They consist in degenerated and necrotic changes in ganglion cells and in proliferative and exudative changes in the interstitial tissue. The changes are confined largely to the proximal end and to one side of the ganglion. In the affected areas the capillaries and small veins are dilated, and here and there small areas of hemorrhage are seen. The degenerative changes of the ganglion cells vary from swelling and cloudiness, with failure of the granules to stain, to hyaline change and complete necrosis. Some cells show larger and smaller vacuoles. The necrotic cells are shrunken, and their nuclei do not stain. The nuclei of some of the degenerated cells stain diffusely. There is proliferation of the cells of the spaces about the ganglion cells. The latter are not, however, invaded by cells. The right ganglion is normal.

Posterior Nerve Root. Left. The inflammatory changes here are extreme. The nerve is surrounded by a thick fibrinopurulent exudate, and in many places there are larger and smaller areas of purulent exudation in the nerve itself. This is associated with both hemorrhages and proliferation of nerve cells. Similar but less intense changes are found in the right posterior root.

The Fourth Cervical Ganglia and Nerve Root. The left ganglia and its root show the same marked degenerated and inflammatory changes described in the third left. The right ganglia is normal, but its root shows marked perineuritis and endoneuritis. The other ganglia are normal.

Spinal Cord and Meninges. Sections of the cord and meninges cut at various levels show meningeal inflammation in varying degree. They are most marked at the level of the third and fourth cervical ganglia. Here the pia arachnoid is the seat of a marked fibrinopurulent exudation, which extends along the nerve roots on both sides. The polymorphonuclear neutrophilic leukocyte is the most common cell in the exudate, but large and small mononuclear leukocytes are also present. There are also numbers of large cells with single oval nuclei. A few long, thread-like organisms are found.

The cord shows no inflammatory changes, and its ganglion cells appear normal.

Anatomical Diagnosis. Actinomycotic abscess of the right occipital lobe rupturing into the right lateral ventricle. Suppurative

inflammation of the sphenoidal sinuses, acute seropurulent cerebrospinal meningitis. Acute neuritis of the fifth nerve and of the third and fourth and other posterior nerve roots. Acute inflammation of the third and fourth posterior root ganglia of the left side; herpes of the left side of the back of the neck and of the left scapular region. Slight actinomycotic bronchopneumonia.

SUMMARY. A review of these cases shows a complete correspondence between the situation of the herpetic lesions and the ganglionic changes. In each case the distribution of the herpes was limited to areas of skin or mucous membrane supplied by the ganglia, which were the seat of the degenerative inflammatory lesions. In those cases with herpes of areas supplied by nerves from the Gasserian ganglion this relation is even more precise. In Case I., for instance, with herpes on the nose near the inner canthus of the eye, the ganglionic lesion was limited to the point of origin of the superior maxillary nerve. In Case IV., with multiple herpetic lesions of the head (nose, upper lip, cheek, hard and soft palate), the lesions of the Gasserian ganglia again corresponded completely to the portion giving origin to the nerves supplying these parts. In Cases II. and III., though unsupported by anatomical examination, in view of the findings in the other cases, the inference that the herpes was associated with lesions of the Gasserian ganglia seems justified.

Case V. is of particular interest, for it demonstrates that, just as in spinal meningitis with involvement of the nerve roots, herpes does not occur unless the inflammatory changes extend to the ganglia; so in marked neuritis of the sensory root of the right fifth nerve, without appreciable changes in the Gasserian ganglion, herpes of the part supplied by this ganglion does not occur. In this case, under these conditions, there was severe trifacial neuralgia without herpes. Degenerative changes, with or without inflammatory lesions of the sensory ganglia, are therefore necessary for the excitation of the nervous impulses upon which the production of the herpetic lesions depend.

Cases IV. and V. also confirm the opinion expressed in my previous paper that the herpes of cerebrospinal meningitis is due to inflammatory lesions of the sensory ganglia.

The cases reported in this article furnish additional proof to support the position that the ordinary herpes zoster, as well as the herpes of pneumonia and cerebrospinal meningitis, is a pathological condition dependent upon definite lesions of certain sensory ganglia, which may be caused by a variety of agents acting in different ways. The herpes is dependent upon the ganglionic lesions, and apparently does not vary with causes of the latter, whether they are due to embolism, thrombosis, hemorrhage, invasion by tumors, microorganisms and other toxins or to other poisons. The ganglionic changes in the cases of herpes complicating pneumonia and cerebro-

spinal meningitis studied by me are identical with those described by Head and Campbell in their acute cases of so-called spontaneous herpes zoster. In the herpes of meningitis the ganglion changes are probably commonly due, as pointed out by Councilman, Mallory, and Wright, to an extension of the inflammatory process along the nerve roots to the ganglia. In pneumonia the matrices morbi are apparently brought to the ganglia by the circulation.

References.

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A CASE OF SYRINGOMYELIA WITH DOUBLE OPTIC NEURITIS.

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AND

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OPTIC neuritis as a complication of syringomyelia is of such unusual occurrence that a report of such a condition even without necropsy should be of interest.

We have been able to find only two similar cases in the literature, one by Bullard and Thomas, reported in this JOURNAL in 1899, and the other by Saxer.[1] Although neither of these instances were diagnosed during life, their clinical histories to a large extent resembles ours. A brief *résumé* of these cases is of interest.

Boy, aged six and one-half years. His family and the past history of the patient were unimportant. At three years of age severe headache and a little later vomiting appeared. These symptoms gradually increased in severity and a year after they began marked double optic neuritis developed, this being followed by optic atrophy. Ten months after the blindness began, a sudden paralysis of the lower limbs appeared, followed by anæsthesia and flexor contractures in the lower limbs and disturbances in the function of the bladder and rectum. The headache, blindness, and the vomiting persisted

[1] Beiträg. z. path. Anat. u. z. allgem. Path., Bd. xx. No. 2.

until death, which was three years after the beginning of the symptoms. The pupils were dilated and did not respond to light or accommodation. There was a paralysis of both external recti. Convergence was normal. A temporary paralysis of the left seventh nerve had been present. Hearing and taste were not affected, and bulbar symptoms were not present.

At the necropsy a cavity was found in the spinal cord extending from the third lumbar segment to the pyramidal decussation. The lateral, third, and fourth ventricles were markedly dilated.

In Saxer's case a girl of sixteen had severe pain and considerable weakness in the lower limbs. There was marked double choked disk with converging strabismus and disturbance of movement, especially in a lateral direction of the left eye. At the necropsy a syringomyelic cavity was found extending to the upper part of the cervical cord, and in the brain a marked congenital internal hydrocephalus was present.

The history of our case is as follows: A girl, aged sixteen years, has been under our observation for the past six months. Her paternal grandmother died of cancer and her paternal grandfather of "paralysis of the brain." Her mother died of hemorrhage one week after the birth of the patient. Has two brothers and one sister living and well. One brother died of fever at the age of seven.

The patient was born at full term in easy labor and was said to be a sickly babe. Had many of the ordinary diseases of childhood, but was comparatively well until the age of seven, when she started to grow exceedingly fast, being now about six feet tall. She would, however, play and behave much like other children until she was twelve years of age.

Mentally she had always been considered peculiar, and was said to have epilepsy when a child. Since the age of twelve she has gradually become stupid and her powers of attention, imitation, and memory have become very poor. She is very indolent, obstinate in some things; is very careless of her clothing, and spends most of her time sitting and staring. Speech is normal, there being entire absence of bulbar phenomena.

Coincident with the appearance of the mental symptoms a decided unsteadiness appeared in her walking, and when first examined several months ago she was barely able to walk with assistance, dragging both legs, especially the left. At present she is chair-fast.

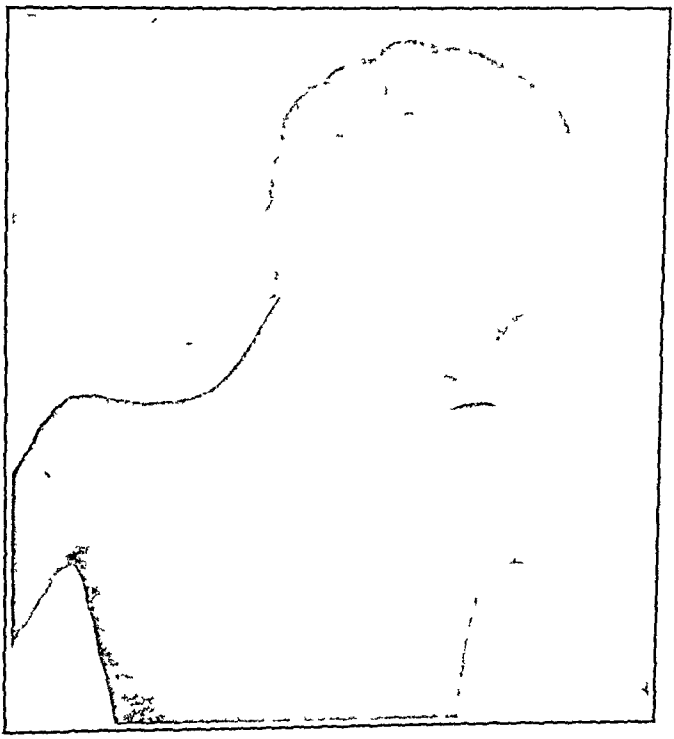
She has been complaining of more or less frontal headache for the past two years, and occasionally vomits forcibly. Has not had nausea, dizziness or convulsions, and when first made to stand did not stagger. The eye symptoms have come on during the past two years until now she is completely blind in her right eye and almost so in the left.

The patient while sitting always inclines her head forward and to the left. There is a lateral spinal curvature to the right in the

dorsal region. She is extremely thin but not emaciated, and there is no particular atrophy anywhere. The skin is dry and scaly.

Movement is possible in all directions in the upper limbs, but they are weak. The grasp of each hand is poor. The bicep reflexes are present in each side but are much diminished, while the triceps and wrist reflexes are absent. There is ataxia of both upper limbs, more marked in the left. The lower limbs are contractured in flexion at the knees. Is able to move her ankles weakly. The patellar and Achilles jerks of each side are much and equally exaggerated, and ankle clonus is occasionally present. The Babinski reflex was not obtained, the toes not moving in plantar irritation.

FIG. 1



Photograph of the patient, illustrating the constant deviation of the head to the left

Sensation for all forms is normal over the face. In irregular areas in the left forearm, arm, shoulder, and chest, both in front and back, touch is properly recognized, but pinprick and both heat and cold are not recognized, or are called touch. Sensation seems normal over the rest of the body.

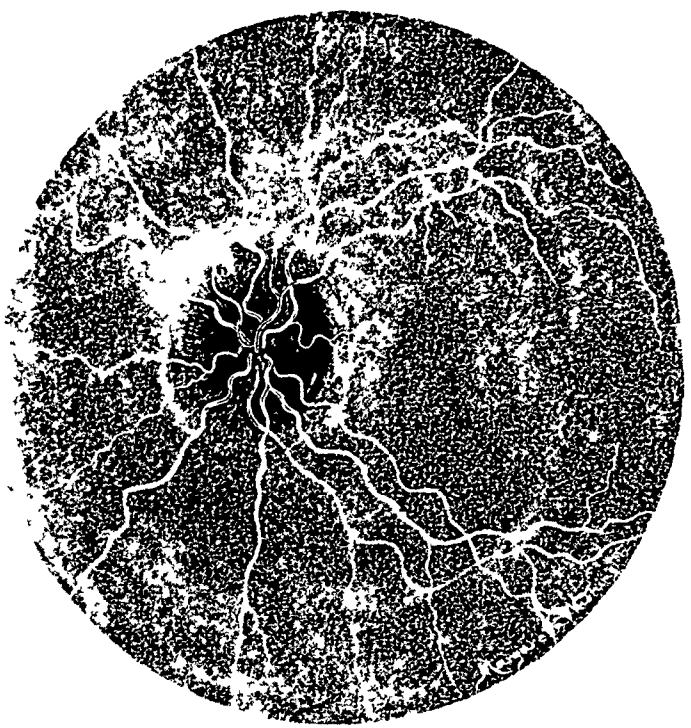
There is incontinence of urine and feces at times, but it is a question whether this is not due to her mental condition.

The tongue is protruded straight, there being no tremor or atrophy. The facial nerves are normal, and the masseters contract well in either side. Hearing seems disturbed on the left side.

The following is Dr. Thorington's report from examinations made March 28, May 2 and 17, 1905: Both eyes under the influ-

ence of atropin. Pupils are dilated equally round, 8 mm. in diameter. Eyeballs are not prominent. There is no ptosis or nystagmus. The vertical width of palpebral fissure in the centre is 8 mm. When either eye is brought to the median line the upper lid covers the upper portion of the cornea to the extent of about 2 mm., and at the same time the sclera is exposed for about 1 mm. in width between the lower edge of the cornea and the upper edge of the lower lid. In a state of repose each eye is decidedly divergent; the temporal edge of each cornea being hidden for 1 or 2 mm. in the outer can-

FIG 2

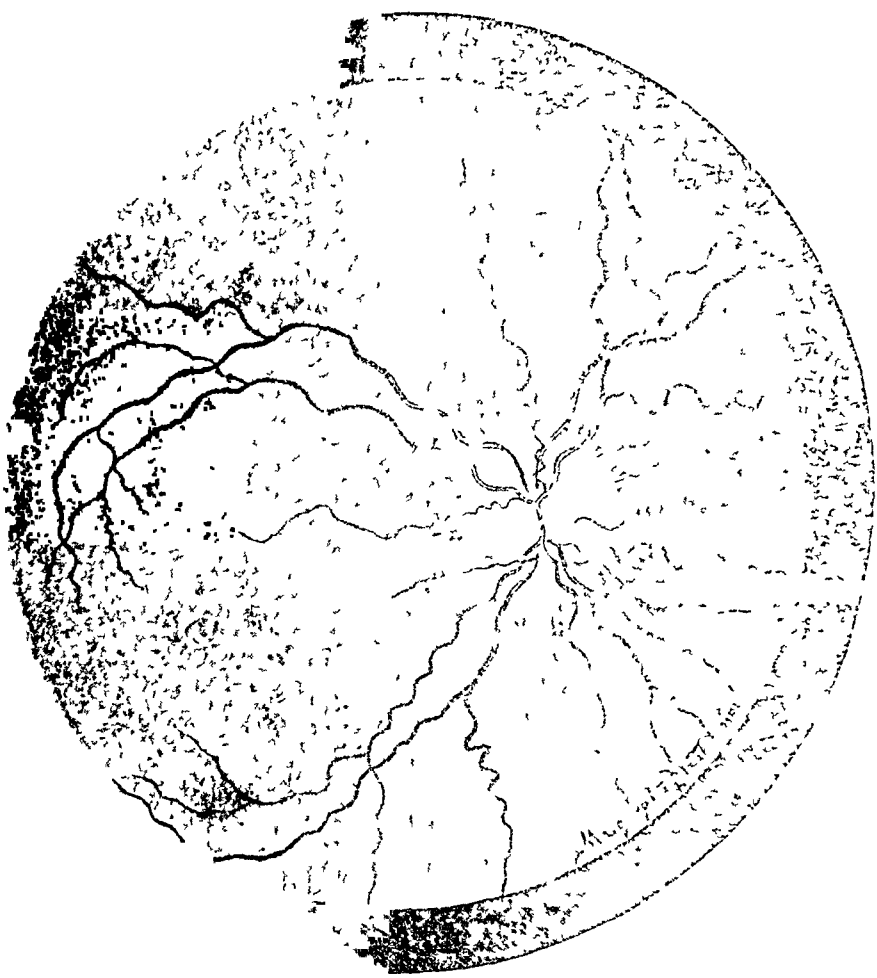


thus. Neither eye will converge or change its position from divergence when the finger is advanced toward the nose in the median line. The tension of each eye is normal, and there are no other gross external changes present. When not under the influence of atropine the pupils are 5 mm. round and the irides respond slightly to light and accommodation, the left a trifle more than the right. There is weakness of both external recti. Associated movement upward and downward is of normal extent, but are weak because she is unable to fix the eyeballs in these directions for any length of time.

Apparently the right eye is blind. The patient states that she cannot see the small five-volt electric light when held close to the eye. Neither does the eye wink unnaturally when the finger is moved slowly or rapidly close to the eye.

The left eye is not blind and the patient is able to count fingers eccentrically at a distance of ten inches. The field of vision is apparently limited to the nasal field, though this is not positive, as the patient's mental condition is too uncertain for definite answers.

FIG. 3



Ophthalmoscopic Examination of the Right Eye. The cornea, lens, and vitreous are clear, no opacities present. Eye-ground "flannel red" in color. There are no hemorrhages present nor are there any indications that there ever had been any hemorrhages. The macula is without any changes and the perimacular region is normal. The disk is quite prominent, round, and its edges striated, hazy, and of a dirty gray color. The central portion of the disk is chalky white in color. The apparent width of the disk as it extends into the surrounding eye-ground is about twice that of the normal

disk. The retinal vessels are much smaller than normal, the veins are not unusually tortuous at the present time and the light streak in the veins and arteries is seen only upon the disk and extending out a very short distance from it. The elevation of the disk is best seen with a plus four diopter lens at the sight hole of the ophthalmoscope and the surrounding eye-ground is seen with a plus two; this gives a swelling of the disk of about two diopters, which would represent about $\frac{2}{3}$ of a mm. The refraction of the eye is therefore two diopters of hypermetropia without any astigmatism.

Ophthalmoscopic Examination of the Left Eye. The findings are the same as in the right eye with the following important additions —viz. the disk is best seen with a plus five diopter lens, and as the refraction is the same as in the other eye, the swelling of the disk is about 1 mm.

There is considerable broken pigmentation in the retina to the temporal side of the disk.

Diagnosis. Well-developed papillitis with beginning postpapillitic atrophy, much more advanced in the right eye, which was presumably the eye primarily involved. The papillitis, which is still present in each eye and well illustrated in the colored sketches by Miss Washington, is apparently an unusual variety of papillitis, and is known as "engorgement" papillitis of von Gräfe in contradistinction to the more common variety of papillitis known as inflammatory. This diagnosis is substantiated by the (*a*) absence of hemorrhages, (*b*) the absence of macular changes (macular figure), (*c*) by the characteristic swelling of the disk which ceases abruptly at the edge. Of course, this diagnosis of "engorgement" papillitis is not always positive, for the two varieties of papillitis are not as widely separated ophthalmoscopically as we might wish, and yet with the diagnosis of syringomyelia there is little if any doubt as to the correctness of this opinion.

Two possible causes can be mentioned to explain the optic neuritis which complicates syringomyelia. First, an intracranial growth, and second, an internal hydrocephalus.

Of course, a tumor almost anywhere in the brain may cause optic neuritis, but neither in the two reported cases nor in our case can this possibility be admitted. Hoffmann suggested that a tumor situated in the medulla oblongata, and which formed the starting point of a syringomyelia, could easily cause optic neuritis. This of course may be so, but in the cases reported by Saxer and Bullard and Thomas the syringomyelic cavity did not extend beyond the lower part of the pyramidal decussation.

More probably as in the cases of Saxer and Bullard and Thomas, internal hydrocephalus caused the optic neuritis in our case. Schlesinger in his admirable monograph on syringomyelia expresses himself of the opinion that hydrocephalus is a frequent complication of syringomyelia, and mentions eleven such cases. He further states

that through pressure caused by the increased amount of cerebrospinal fluid there may be palsy of different cranial nerves. This is undoubtedly the explanation for the paralysis of the external recti muscles and the possible weakness of associated movements present in our case.

CHRONIC ANTERIOR POLIOMYELITIS, WITH THE REPORT OF A CASE WITH NECROPSY.

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CLINICAL NOTES AND REMARKS BY DR. MOLEEN.

Owing to the diversity of opinion with reference to the classification of spinal-cord lesions attended with muscular wasting, with or without spasticity, and with or without the presence of the deep or tendon reflexes, the assigning of the following case presents a somewhat difficult problem, and for this reason it is deemed wise to first detail the clinical notes of the case before discussing those points, which, to the author, were obstacles in arriving at an appropriate clinical diagnosis.

J. Ten W., aged thirty-seven years, laborer, though he did mostly upholstering work, was first seen October 3, 1903, at which time the following notes were made: He was born in St. Clair, Minn., but has been in Colorado six or seven years, and did not come for his health.

Family History. He denies all diseases of childhood, except measles, which he had at the age of ten years. He was perfectly well until 1890, when he had an attack of la grippe, and he had another in Colorado three years ago. His calloused hands bear him out in the statement that he has done hard work, and he says that he has been exposed in the mountains to rough weather until a few years ago, though he denies rheumatism or pneumonia. He admits having contracted gonorrhœa, but firmly denies syphilitic infection. It may be stated, however, that a physician related to him informs me that he treated him for syphilis twenty years ago.

Present History. About two weeks ago, without chill, pain, cold, fever, numbness, or the slightest symptom of an acute character,

he noticed upon arising in the morning that his left foot felt heavy and that he was "limping."

He insists that he was perfectly well at the time and that the weakness was his only cause for complaint. He is certain that he is no worse to-day than when he first noticed his trouble. He has no difficulty in passing his urine nor in emptying his bowel.

FIG. 1



Showing the wasting of the lower limbs and foot-drop.

Examination, October 6, 1903, gave the following results:

He is a short—five feet, three inches—well-developed man, though he shows his age and hard work. His weight is 120 pounds.

In walking the left foot is raised considerably higher than the right, and with a jerky exaggerated flexion of the knee the foot is planted squarely on the floor with a tendency to strike the toe first.

Standing with the eyes open and the feet together, there is no perceptible swaying, which is but slight when the eyes are closed.

In the sitting posture, with lower limbs hanging, the "foot-drop" is noticeable on the left side.

There is no ataxia in the arm nor trunk muscles.

The calf of the left leg is somewhat atrophied and the muscles flaccid, though especially noticeable is the wasting of the muscles supplied by the peroneal nerve. The dorsum of the left foot, from the prominence of its tendons, seems more wasted than the right.

The circumference of the largest part of the calf: right, $12\frac{3}{4}$ inches; left, $11\frac{1}{2}$ inches. Of the thigh: right, $17\frac{1}{2}$ inches; left, $15\frac{3}{4}$ inches.

Electrical reactions: Galvanic battery is out of order and reactions cannot be taken. Faradic irritability is greatly diminished in the lower part of the left leg, otherwise it is normal throughout this limb. With the slowly interrupted current no contraction of the muscles supplied by the peroneal nerve can be obtained, even with the strongest current.

Reflexes: Knee-jerks, right is increased, especially on reinforcement. Left is slightly more increased than the right, but the knee-jerks were not increased to the degree commonly observed in spastic paralysis. Ankle clonus is absent. Tendo Achillis reflex: Right is present and about normal. Left is absent.

Deep reflexes of the forearm, flexors and extensors: Right are present. Left also and slightly increased. Biceps, triceps, and deltoid, same. Dynamometer (Tiemann, inner scale): Right = 280; left = 280. The man is right-handed. Masseter reflex is absent.

Superficial reflexes: Plantar: right is present; left is very slight. There is no evidence of the Babinski phenomenon. Cremasteric reflex: Right is present; left also is present, though not so marked as right. Lower abdominal reflex: Right is slight; left is very slight. Epigastric reflex is present.

Sensory phenomena are normal throughout the body (tactile pain, pressure, temperature, and postural senses taken).

Special senses: Eyes, all external ocular motion is normal and without nystagmus. Pupils are equal and respond actively to light and accommodation. Fields of vision are apparently normal. Fundi: There are no retinal changes, and the disk is well outlined in either eye. Hearing (watch): Right = 6–24; left = 8–24. The tuning-fork is heard best in the closed ear, and aerial conduction is greater than that of bone. Taste and smell are seemingly not impaired. The tongue is protruded in the median line, without tremor, and there is no difficulty in speech.

A diagnosis of anterior poliomyelitis was made at this time and he was under observation until January, 1904. During this time there was no apparent change in his condition, and it may be said that, though repeatedly examined, there was no wasting to be noticed in the hand nor in any other part of the body, nor was

fibrillation to be seen at any time, these symptoms having been expected because of the absence of improvement.

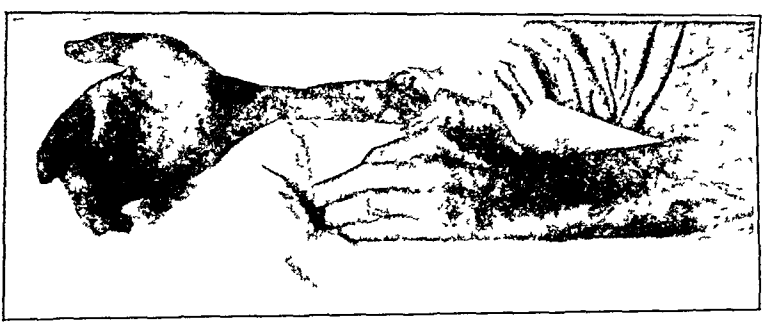
He was not seen again until January 21, 1905, when the following notes were taken:

The face is apparently drawn, probably due to emaciation, and moderate emotion (laughter) is not evidenced in the facial expression. All voluntary motion of the facial muscles, though weak, is equal on the two sides. The tongue is protruded about half an inch beyond the incisor teeth, shows a decided fibrillary tremor throughout, and is markedly furrowed from wasting. It is protruded in the median line.

He responds correctly to questions asked him, though with very slow enunciation, drolling all words requiring the use of the pharyngeal and labial muscles, but more particularly the lingual.

He states that six weeks ago he "had his palate cut" (referring to the uvula), from which time he noticed difficulty in swallowing and speaking.

FIG 2



Showing the wasting of the hands and forearms

There is to be noticed in the most prominent muscles—*e. g.*, biceps, deltoid, pectoral, gluteal, etc., linear depressions parallel to the muscle fibres, apparently wasted strands, between which lines of twitching are to be seen. This twitching is also to be plainly seen in the spinal muscles. Tapping the muscles increased the frequency though not the degree of the twitching.

The greatest wasting is to be observed in the hand muscles on both sides, more particularly the adductors of the thumb. The thenar and hypothenar eminences are completely wasted, and on extending the hands a decided, somewhat coarse tremor of the right thumb is noticeable (Fig. 2).

The left lower extremity is smaller in size than the right, and the group of muscles innervated by the left peroneal nerve is entirely wanting.

Plantar flexion of the left foot is somewhat weaker than of the right, while no dorsoflexion of the left foot can be accomplished.

The muscular strength in the shoulder, arms, and forearms is fairly well preserved and equal. Dynamometer, right = 110; left, = 100.

The man walks raising the left knee markedly higher than the right, and the left foot is swung farther forward and placed flatly on the floor and turned slightly outward. His gait is little if at all altered upon closing the eyes. He is able to stand with the feet together and eyes open, though he sways slightly, and the swaying is somewhat increased when the eyes are closed. The increased effort in the latter instance seemingly excites a fine tremor of the face and neck muscles.

The arm and trunk muscles are free from ataxia.

Measurements of the extremities: Arms, 4 inches above the olecranon; right = $8\frac{3}{4}$ inches; left = $8\frac{1}{4}$ inches. Arms, 4 inches below the olecranon; right = 8 inches; left = $7\frac{1}{2}$ inches. Legs, 4 inches below tibial tuberosity; right = $10\frac{3}{4}$ inches; left = $9\frac{1}{2}$ inches. Thighs, 8 inches below anterior superior spine, right = 15 inches; left = $12\frac{1}{2}$ inches.

Electrical reactions: galvanic (chloride of silver fifty-cell battery was used, sponges moistened in alkaline tepid water, and interruptions made with the handle; current strength in milliampères):

Tibialis anticus: right, KaClC 10, KaOC 10, AnClC none, AnOC none; left, no response to the strongest current; peronei same.

Tibialis posticus: right, KaClC 6, KaOC 12, AnClC 10, AnOC none; left, KaClC 2, KaOC 3, AnClC 5, AnOC 20.

Flexor carpi radialis: right, KaClC 2, KaOC 15, AnClC 6, AnOC 25; left, KaClC 2, KaOC 20, AnClC 3, AnOC 15.

Extensor communis digitorum: right, KaClC 4, AnClC 3; left, KaClC 4, AnClC 2 (weaken rapidly).

Both the right and left deltoid muscles give anodal and cathodal contractions at 3 milliampères.

Masseters are equal: KaClC $1\frac{1}{2}$, AnClC $3\frac{1}{2}$. Sternocleidomastoid muscles are equal: KaClC 2, AnClC 3.

There can be no responses obtained from the thumb muscles of either hand with the strongest currents.

Faradic: (No. 8 chloride of silver battery, No. 2 secondary coil, 26 wire; two cells in circuit and current strength indicated in fractions of the rheostat circumference, unless otherwise indicated).

Tibialis anticus: Negative, right, $\frac{1}{3}$; left, none with full current. Positive, right, $\frac{1}{2}$; left, none with full current.

Rapid interruptions the same.

Tibialis posticus: Right, $\frac{1}{2}$; left, $\frac{1}{2}$ (negative).

Extensors of both legs respond equally to $\frac{1}{2}$; flexors, $\frac{3}{4}$.

Flexor communis digitorum: left, $\frac{1}{3}$; right, same.

The muscles of both arms and forearms show an increased irritability. Facial muscles are about normal, electrically.

Reflexes: Knee-jerks, right, increased; left, also. The effect is little changed by reinforcement. A slight ankle clonus of short duration is present in the right foot, which is very easily exhausted. Left absent. Deep reflexes of the forearm: Flexors and extensors present, those of the left side are seemingly more pronounced than those of the right side. Biceps, triceps, deltoid, and pectoral reflexes are present. Masseter reflex is absent.

Superficial reflexes: Plantar, right is present; left is absent. There is no evidence of the Babinski phenomenon, and the dorsal foot reflex lately described by Mendel[1] is negative. Cremasteric reflex: right is slight; left is absent. Lower abdominal reflex apparently is absent. Epigastric reflex is slight.

All sensory phenomena (tactile, temperature, pain, pressure, and postural) are normal and equal.

Owing to lack of control of the tongue, it is difficult to accurately test the sense of taste, though it is seemingly normal, as is also the sense of smell.

Eyes: Pupils are equal and respond equally to light and accommodation. Ocular motion is equal in all directions. The fields of vision (taken with the hand) are evidently not contracted. The disks, while pale, show no degeneration and the vessels are not changed.

Hearing is about as previously recorded.

The foregoing notes were taken during several examinations made between January 21 and 29, 1905, to which the following general observations may be added: Inability to control emotion; the extremes only were evident in the facial expression; slight laughter was often followed by tears without provocation; increasing dysphagia and dysarthria; increasing difficulty in coughing and expectorating apparently due to lack of control of the respiratory, pharyngeal, and lingual muscles.

When seen January 29, 1905, at 1 P.M., the temperature was 99.5° and pulse 100, and he was sleeping quietly. Refusing nourishment, he slept the greater part of the afternoon, death taking place at 7 P.M. of the same day, the increase in pulse and respiration rate and volume being the only change noticed, the attendants are certain, however, that no marked changes in respiration (*e. g.*, laryngeal spasm, Cheyne-Stokes breathing) were to be noticed.

Autopsy was made three hours after death. The examination was limited to the spinal cord and brain. The body was quite warm. The spinal cord and brain were removed, the cerebellum, brain stem and cord being retained for examination. The membranes were not adherent, no vascular change was noticed, and the ventricles were apparently normal. No gross changes were observed in the cerebral hemispheres. The retained portions were submitted to Dr. William G. Spiller for examination.

[1] K Mendel Neurologisches Centralblatt, 1904, v xxiii No 5

SUMMARY. A man, aged thirty-seven years, probably syphilitic, is afflicted with sudden paralysis of the group of muscles innervated by the left peroneal nerve; complete loss of faradic irritability followed in a week, and this was followed by rapid wasting and slight diminution in size of the entire leg; the deep reflexes were present and increased, but not excessively, and in that spinal segment represented by the absolute atrophy they were lost; the symptoms were confined to the left lower extremity and were without change during three months; no fibrillary tremor, sensory disturbance, hand weaknesses, nor cranial nerve symptoms were present. One year later there were slight general emaciation, complete wasting of the thenar and hypothenar eminences, and, to a less extent, of the interossei muscles; atrophy and paresis of the tongue and pharynx, fibrillation throughout the body, including the tongue, and tendon reflexes still moderately increased. There were no marked electrical changes, except in absolutely wasted muscles, and no bladder, bowel, nor sensory disturbances. He was becoming more emotional, dysphagic and without dyspnœa, though he had a rapidly increasing pulse and respiration. Termination was death.

When first observed the recording of the case as acute anterior poliomyelitis, in view of the rapid onset and the absence of evidence of involvement of other portions of the cord, was justifiable. The only points apparently opposed being the age of the patient and the absence of prodromes or febrile disturbance.

The fact that no evidences of extension nor involvement elsewhere could be noticed after three months seemingly supported this diagnosis.

When, however, wasting of the hands, fibrillation, dysarthria, and dysphagia were added after the lapse of a year, one would be justly suspicious of (*a*) progressive spinal muscular atrophy (especially a peroneal type), (*b*) amyotrophic lateral sclerosis, or (*c*) chronic poliomyelitis—three conditions which have been considered by some authorities as synonymous—the extremely wide variations in individual cases notwithstanding.

It seems pretty well established that in pure cases of progressive spinal muscular atrophy the wasting invariably precedes the paralysis, and certainly the great majority of cases first manifest weakness in the hands (nine-tenths, according to Gowers). Conspicuous also is the advent of fibrillary twitching in the gradually weakening muscles, as well as in those about to be attacked, in which the weakness had not been noticed, and in any event before paralysis had supervened.

The electrical reactions in these cases of progressive spinal muscular atrophy persist until the wasting is intense.

Clinically, perhaps, this case more closely conforms to the type known as "peroneal," which since the thesis of Dr. Tooth and the observations of Dr. Hoffmann, has, in the opinion of most author-

ities, merited a separate classification, and some have referred to it as "neuritic" (perhaps more properly "neurotic," from the "Progressive Neurotische Muskelatrophie" of Hoffmann), in view of its occurrence in several members of the same family, in the majority of instances. That it occurs, as a rule, in the second half of childhood—*i. e.*, before the twentieth year of life, seems to be generally accepted, though Oppenheim thinks the commencement may be as late as the fourth decade.

Some observers have used the term "neuritic" advisedly, because of the belief that the peripheral nerves are primarily affected. This is questioned by Dana, who believes the cord to be first attacked, and quotes Marinesco as having recently confirmed this view, mentioning a citation by the same author of a case beginning as a "typical acute anterior poliomyelitis."

As rather against than for the placing of this case under this heading, the following features deserve mention: The occurrence of the disease earlier in life as a rule; usually hereditary involvement; frequent sensory disturbances (pain, numbness, etc.); usually abolition of the tendon reflexes in the extremity affected; slow development; atrophy usually extreme; lastly, conspicuous by its absence in the cases reported is the mention of bulbar symptoms.

As tersely expressed by Dana, the hereditary or "leg type" of progressive muscular atrophy is characterized by its beginning in the legs, by much sensory disturbance, typical degeneration reactions and hereditary or family history.

Is this a case of amyotrophic lateral sclerosis?

It is well before considering this condition to disregard the question of synonymy, and with Oppenheim, Dana, Beevor, and others, return to the original distinctive classification of Charcot. This disease occurs most frequently after thirty, women being more often affected than men; in true types the hands are first affected, and the spasticity is marked; the first symptom is usually rigidity; clonuses, including a masseter clonus, are frequent; flaccid paralysis never inaugurates the disease, and the onset is nearly always gradual. Oppenheim only records an acute beginning; as a rule, each muscle wastes very gradually.

In the study particularly of the clinical side of the case, however strong our convictions may lead us to the heading of progressive muscular atrophy, the consideration of the distinctive points of the third of the "trio," chronic anterior poliomyelitis cannot be overlooked. This line of distinction, one must admit, in the light of our present knowledge, is as yet fine.

While partial recovery is the rule, though more especially in the acute form of anterior poliomyelitis, yet cases do become subacute or chronic, and extension or probably the independent involvement of other segments, including the bulb, are then not uncommon, and in fact bulbar paralysis very frequently closes the scene in all three

of the conditions mentioned—viz., progressive spinal muscular atrophy, amyotrophic lateral sclerosis, and chronic anterior poliomyelitis.

In chronic anterior poliomyelitis the paralysis precedes the wasting, the palsy is absolute, flaccid, without rigidity, and involves groups of muscles physiologically related, while in the other affections previously considered the muscles concerned are anatomically related. Syphilis is a frequent cause in advanced life of the chronic cases; all electrical irritability is rapidly lost in the group of muscles affected, and neighboring muscles are altered in proportion to the degree of subacute extension, this alteration becoming less marked farthest from the cord representation of the principal group involved. A predilection of the peroneal group in all cases of anterior poliomyelitis is notable.

The mention of fibrillary twitching has been purposely omitted heretofore, for the reason that it is seemingly common to all chronic muscular atrophies, and Gowers, in his broad view of the three diseases as one, states that it is not invariable, nor is it confined to this disease; and Oppenheim has to say: "Progressive muscular atrophy can never be diagnosed from fibrillary tremor, as this symptom may occur in healthy persons after excesses, in emaciated individuals when exposed to cold, and in neurasthenic and hypochondriac patients."

While disposed to rest the diagnosis here, I am constrained to admit that against this disposition is the presence or increased activity of the deep reflexes which in the cases previously reported as such was absent; the explanation offered with reference to the electrical alterations in the segments immediately adjacent to the actual lesion, it is reasonable to suppose, will hold good here, and possibly establish a point of distinction with future observation.

It is therefore evident that while no resting place is absolutely fitted for the reception of this particular case, the wide latitude of which chronic anterior poliomyelitis admits, and the fact that here are found fewer opposing factors than in the other mentioned conditions render its placing here at least permissible.

The emotional symptoms at the close of the case are, as noted by Dana, common evidences of bulbar lesions, either primary or secondary.

PATHOLOGICAL NOTES AND REMARKS BY DR. SPILLER.

The case which Dr. Moleen has studied clinically so carefully is worthy of the attention he has given to it. I know of no case with necropsy like it in American or English literature, and it should, therefore, be of value in the study of the muscular atrophies. This is a subject that is still causing much difficulty in classification. The commencement of the symptoms in Dr. Moleen's patient with

peroneal palsy, resembling the neurotic muscular atrophy, is characteristic of certain lesions of the cells of the anterior horns. Baumann,[1] in a clinical study of 85 cases of acute anterior poliomyelitis, has shown that the muscular supply of the peroneal nerve is especially liable to become paralyzed in this disease.

Dejerine[2] reviews the opinions held regarding motor localization within the spinal cord. According to him, it is not segmental (hand, forearm, arm), as Van Gehuchten and De Buck believed; it is not diffuse (Marinesco), each muscle is not separately represented (Sano), but the localization is of the root type. An examination of the diagram given by Dejerine, p. 780, shows that the peroneal supply is represented in the fourth and fifth lumbar and first sacral segments. We can, therefore, understand why, in Dr. Moleen's patient, the paralysis began in the peroneal group.

My report is as follows:

Only the spinal cord and brain stem of this case were sent to me for examination.

Lumbar Region. The white matter by the Weigert hæmatoxylin stain does not appear to be degenerated. The small vessels of the anterior horns are much congested and are, therefore, very prominent by this stain. Those of the posterior horns are also congested, but not so much so as are the vessels of the anterior horns. Small hemorrhages are numerous in the gray matter, especially in the anterior horns, but are also present in the posterior horns. The posterior roots are well stained. The anterior roots of the lumbar and sacral regions show in places distinct degeneration, but these roots are far better preserved than are the cells of the anterior horns. There is no round-cell infiltration within the spinal cord or pia. The nerve cells of the anterior horns are much diminished in number, but those that still persist appear to be almost normal, and only occasionally one is seen showing chromatolysis, displacement of the nucleus, or much pigmentation. A slight recent degeneration in the crossed pyramidal tracts is seen *only* by the Marchi method.

Middle Thoracic Region. Small hemorrhages are numerous in the gray matter, in the posterior as well as in the anterior horns, and are found, but much less numerously, in the white matter near the gray. The Marchi method reveals a very slight degeneration of the crossed pyramidal tracts and a few scattered black dots elsewhere in the anterolateral columns.

First Thoracic and Eighth Cervical Segments. The small hemorrhages are very numerous in the anterior and posterior horns, and are found also in the white matter near the gray. In this portion of the cord the nerve cells of the anterior horns have almost disappeared. There is hemorrhage also within the pia, but nowhere is there any round-cell infiltration.

[1] Monatsschrift f. Psychiatrie und Neurologie, June, 1905, p. 485.

[2] Sémiologie du système nerveux, p. 789.

Medulla Oblongata. Small hemorrhages are numerous here also. Intramedullary fibres of the twelfth nerve have disappeared by degeneration. Unfortunately the nuclei of the twelfth nerves have been injured at the time of the necropsy, but a marked disappearance of nerve cells in these nuclei may be determined. The nucleus ambiguus contains few cells. The intramedullary fibres of the ninth and tenth nerves are much atrophied. A very slight degeneration of the anterior pyramids is seen only by the Marchi method

FIG. 3



Showing the numerous hemorrhages within the gray matter of the spinal cord.

and is less than the degeneration of the crossed pyramidal tracts of the spinal cord.

There are few cells in the nuclei of the seventh nerves and the intramedullary fibres of these nerves are much degenerated. Small hemorrhages are not so numerous in the pons. The cells still persisting in the facial nuclei stain fairly well, except that here and there a cell may be found distinctly degenerated.

A few small hemorrhages are found in the cerebral peduncles.

The important alterations in this case are disappearance of many of the nerve cells of the anterior horns of the spinal cord and motor cranial nerves in the medulla oblongata, of the anterior roots of the lumbar and sacral regions (cervical region was injured), and of the motor nerves of the medulla oblongata, and numerous small hemorrhages in the gray matter, especially in that of the spinal cord.

It is very questionable whether we should make sharp distinctions between chronic anterior poliomyelitis and progressive spinal muscular atrophy. In the first the paralysis is supposed to develop within a few days or a few weeks, the atrophy appears later, and entire muscles or groups of muscles are paralyzed, the course of the disease is more rapid, and the paralysis develops first in the muscles of the lower limbs or shoulders. In the progressive spinal muscular atrophy the paralysis is proportionate to the atrophy, one muscle fibre after another is affected, and the course is longer than in the chronic poliomyelitis. These are distinctions that are not always regarded, and it is very evident that some of the reported cases of so-called chronic anterior poliomyelitis might just as well be labelled as examples of progressive spinal muscular atrophy.

The cases of either type with necropsy are very rare. To Oppenheim the credit of reporting the first typical case of chronic poliomyelitis with necropsy has been justly given. In Oppenheim's[1] first case the weakness began in the right arm. The vessels of the cord were not distinctly altered.

In the case that S. G. Webber[2] reported in 1875 weakness began in the left knee and soon the left leg gave out. Atrophy did not appear until after the weakness. Sensation was not affected. The nerve cells of the cervical region were much diseased and were not numerous. There was some perivascular bulbar infiltration. The white matter of the cord appeared as a homogeneous mass, not colored by carmine; occasionally an axis-cylinder could be seen. The lumbar and thoracic portions of the cord could not be satisfactorily studied. The nerve cells of the hypoglossal nuclei were few in number and many axis-cylinders were swollen. The posterior and anterior roots were also affected.

This case is important, as it was reported about thirty years ago, and clinically seems to have presented the symptoms of chronic anterior poliomyelitis, but pathologically it appears to have been a case of diffuse myelitis. The duration of the symptoms was about one year.

It is true that Nonne[3] included as cases of subacute anterior poliomyelitis those reported by Eisenlohr, and Landouzy and Dejerine, and as a case of chronic anterior poliomyelitis the case

[1] Archiv f. Psychiatrie, 1888, Bd. xix. p. 381.
[2] Transactions of the American Neurological Association, 1875, p. 55.
[3] Deutsche Zeitschrift f. Nervenheilkunde, 1891, Bd. i. p. 186.

of Dreschfeld. These three cases he acknowledges were very different from one another, and he makes three types of chronic anterior poliomyelitis: 1. The circumscribed form (Eisenlohr's case), in which the lesion is confined to a limited area. 2. A form in which paralysis and atrophy *en masse* occur quickly, show a tendency to arrest or to complete restoration, and may possibly be the result of acute or chronic infection (Landouzy and Dejerine's case). 3. A form which progresses slowly and irresistibly with atrophy of nerve cells without secondary atrophy of peripheral nerves (Dreschfeld's case) or with this atrophy (Oppenheim's case).

Oppenheim, in a later paper, does not accept as cases of chronic or subacute anterior poliomyelitis those referred to by Nonne, and it seems to me that Oppenheim is right in this opinion.

Eisenlohr's case[1] was remarkable on account of the limitation of the process. The weakness and atrophy affected only the right upper limb and persisted about five years. The nerve cells were degenerated only in the right anterior horn between the sixth cervical segment and the first thoracic. The white matter was normal. It is difficult to decide why this case was regarded as one of subacute anterior poliomyelitis and not as one of progressive spinal muscular atrophy.

Dreschfeld[2] regarded the case he reported as one of chronic anterior poliomyelitis. Weakness began in the right foot and leg. The tendon reflexes of the upper limbs were very prompt. The nerve cells of the anterior horns were much degenerated. Lateral sclerosis was found in the cervical region. It seems doubtful whether this should be regarded as a case of amyotrophic lateral sclerosis or as one of chronic anterior poliomyelitis.

Landouzy and Dejerine[3] did not regard their case as one of chronic anterior poliomyelitis.

A case that probably should be classed as one of chronic anterior poliomyelitis with necropsy was reported by Richard Ewald.[4]

The case that Nonne reports began with weakness in the left shoulder and left upper arm. Atrophy occurred at the same time as the paralysis or followed soon after. In the last months of the disease pains were felt in the upper part of the upper limbs and in the thighs. Nonne[5] found some degeneration of peripheral nerves, as well as of nerve cells, in the spinal cord, and he refers to the fact that in Oppenheim's case the hypoglossal and spinal accessory nerves and the nerves within muscles were not entirely normal. Nonne found atrophy of nerve fibres in the white matter of the cord, in the pyramidal tracts as well as elsewhere, but not as symmetrical degeneration.

[1] Neurologisches Centralblatt, 1882, p. 409.

[2] Brain, 1886, p. 173.

[3] Revue de méd., 1882.

[4] Ein Fall von Poliomyelitis anterior chronica, Inaugural Dissertation, Marburg, 1889.

[5] Deutsche Zeitschrift f. Nervenheilkunde, 1891, Bd. i. p. 136.

In Darkschewitsch's[1] case weakness and atrophy developed gradually in the muscles of the upper limbs, neck, back, and partly in the lower limbs. The nerve cells of the anterior horns of the cervical and thoracic regions were diminished in number, and there were numerous hemorrhages which seem to have been in the gray matter of the cord. Rarefaction of the lateral and posterior columns was observed. It is difficult to determine from the brief report why this was regarded as a case of chronic poliomyelitis and not as one of progressive spinal muscular atrophy.

In Oppenheim's[2] second case of chronic anterior poliomyelitis the weakness began in the right shoulder and right upper arm, and after a year extended to the corresponding parts of the left upper limb. Atrophy soon followed the weakness. Paræsthesia was felt in the fingers in the beginning of the disease. The nerve cells of the anterior horns of the cervical and thoracic regions were much degenerated. The columns of Burdach were also degenerated.

In the case reported by A. Dutil and J. B. Charcot,[3] the symptoms began with weakness in the upper limbs. The vessels of the cord were thickened and some small recent hemorrhages were found in the cord. This is the same case that J. B. Charcot later reported alone.

In J. B. Charcot's[4] two cases of chronic anterior poliomyelitis there was some degeneration of the anterior fundamental fasciculus—*i. e.*, of the fibres near the anterior horn. These cases possibly might be regarded as examples of progressive spinal muscular atrophy.

It is difficult two understand why Dejerine's[5] two cases should be regarded as examples of chronic anterior poliomyelitis and not of progressive spinal muscular atrophy, if a sharp distinction is maintained. In one case there was a questionable sclerosis of the interior portion of the right lateral column.

Nonne,[6] in 1896, recognized as cases of chronic anterior poliomyelitis with necropsy the two cases of Oppenheim, the two cases of his own, the case of Darkschewitsch, the case of Dutil and J. B. Charcot, and the two cases of Dejerine.

In Nonne's second case the weakness began in the shoulder muscles. The nerve cells of the anterior horns were much degenerated and rarefaction of the white matter was seen in the anterior, lateral, and posterior columns. The bloodvessels were not distinctly altered. He attributed the poliomyelitis in this case to diabetes. Nonne refers to the slight changes in the white matter observed in Oppenheim's two cases, in his own two cases, in

1 Neurologisches Centralblatt, 1892, Bd. xi. p. 221.
2 Archiv f. Psychiatrie, 1892, Bd. xxiv. p. 822.
3 Le progrès médical, March 17, 1894, p. 185.
4 Archives de médecine expérimentale, 1895, vol. vii. p. 441.
5 Comptes-rendus heb. des séances de la Soc. de biologie, 1895, p. 188.
6 Berliner klin. Wochenschrift, March 9, 1896, p. 207.

Darkschewitsch's case, in Dutil and Charcot's case, and in one of Dejerine's cases.

In P. Schuster's[1] atypical case of chronic anterior poliomyelitis, which was complicated by degeneration of the posterior roots and of the columns of Burdach, the patellar reflexes were much exaggerated. The explanation was supposed to be found in the slight alteration of the lateral columns.

Max Bielschowsky[2] says there are very few cases of chronic anterior poliomyelitis reported that show evidences of inflammation. In Bielschowsky's case the weakness began in the lower limbs, in the peroneal distribution, as in the case Dr. Moleen and I report. He says that contrary to the diagnosis of neural atrophy (peroneal type) were the later developments of the disease and the absence of subjective and objective disturbance of sensation. Contrary to progressive spinal muscular atrophy were the location of the atrophy, the relatively rapid development of the symptoms, and the early loss of function in the diseased muscles. Hemorrhages were found in Bielschowsky's case in the anterior horns from the lumbar to the upper cervical region. In some places the red blood corpuscles had disintegrated, blood pigment was present, and reactive processes had occurred about some of the hemorrhages. These hemorrhages were regarded as evidences of inflammation. Slight degeneration was seen in the crossed pyramidal tracts of the spinal cord as high as the pons, but was detected only by the Marchi method and was slight, exactly as I have found in the specimens studied by me. Bielschowsky observed slight perivascular round-cell infiltration and newly formed bloodvessels. The hypoglossus nuclei also were affected. This case resembles ours very closely, although the disease in Bielschowsky's case lasted nine years. The tongue was also weak and atrophied. No statement is made concerning the condition of the patellar reflex until late in the disease, when the atrophy was intense.

In Grunow's[3] case the symptoms seem to have existed about nine months. The weakness began in the right foot and peroneal palsy developed. Pain was felt in the shoulders and back. Progressive spinal muscular atrophy was excluded because the weakness began in a lower limb, because of the wide extent of the process, the implication of intercostal muscles, and the relative rapidity of development. As in our case, peroneal palsy was among the earliest signs of the disease. The pain in this case is noteworthy. Cellular infiltration was found about the arteries of the anterior fissure and about some of the vessels of the anterior horns and white matter. Necrotic foci were present in the crossed pyramidal tracts, and there were some small hemorrhages in the gray matter of the cord.

[1] Neurologisches Centralblatt, 1897, Bd. xvi. pp. 342, 392, 449.
[2] Zeitschrift f. klin. Med., 1899, Bd. xxxvii. p. 1.
[3] Deutsche Zeitschrift f. Nervenheilkunde, 1901, Bd. xx. p. 533.

The nerve cells of the anterior horns were much degenerated. Degeneration was found in the anterior and lateral columns.

Bruining[1] has observed chronic anterior poliomyelitis in father and son, and in one of the cases obtained a necropsy. In neither case were there periods of abrupt increase in the symptoms. The weakness seemed to be proportional to the atrophy. In the father the weakness and atrophy began in the right thigh and then the shoulders became implicated. In the son the atrophy began in the muscles of the neck and extended to the shoulders and upper limbs. The nerve cells of the anterior horns throughout the cord were degenerated, but no signs of inflammation were found. The capillaries of the cord were congested and in some places blood corpuscles were found in the tissues about the vessels. Bruining is not inclined to make sharp distinctions between chronic anterior poliomyelitis and progressive spinal muscular atrophy.

Rossolimo,[2] in reporting a case of chronic anterior poliomyelitis with syringomyelia, recognized that somewhat exaggerated patellar reflexes may occur in anterior poliomyelitis, and refers to the cases of Darkschewitsch and Schuster. He recognizes as cases of chronic anterior poliomyelitis only those of Oppenheim, Nonne, Darkschewitsch, Schuster (not a pure case), Bielschowsky, and R. Ewald.

Oppenheim, in speaking of chronic anterior poliomyelitis in his text-book, says exaggeration of tendon reflexes alone is not sufficient to make the case one of amyotrophic lateral sclerosis, as exaggeration of reflexes may occur in neurasthenia.

In the monograph on poliomyelitis written by Elis Lövegren,[3] the author reports a case of subacute poliomyelitis with necropsy. The weakness began in the fingers and was followed in two or three months by atrophy of the hands. The paresis and atrophy became widespread. The patellar reflexes were exaggerated. The nerve cells of the anterior horns of the spinal cord and of nuclei of motor cranial nerves were degenerated. Vessels with thickened walls were found in the spinal cord, but there was no round-cell infiltration. The white matter of the cord was said not to be involved and the pyramidal tracts to be intact. The method of Marchi was not employed. The anterior roots and peripheral nerves showed slight atrophy. A cavity in a limited area was found in one anterior horn.

Lövegren refers to two cases of subacute or chronic poliomyelitis with necropsy reported by Philippe and Cestan,[4] in which a slight sclerosis of the anterolateral columns was found; and to a case with necropsy reported by Raymond and Philippe.[5] I have not been able to obtain the paper by Philippe and Cestan.

[1] Deutsche Zeitschrift f Nervenheilkunde, 1901, Bd. xxvii. p 269

[2] Neurologisches Centralblatt, 1903, p 388

[3] Sonder Abdruck a d. Jahrbuch f Kinderheilkunde, S. Karger, Berlin, 1905, vol. lxi

[4] XIII Congrès international de médecine, Paris, 1900, comptes rendus, section de neurologie, p 271.

[5] Soc. de neurologie, Nov. 6, 1902, Archives de neurologie, 1902, 2 série, tome xiv. p. 537.

The case of chronic poliomyelitis very briefly reported by Raymond and Philippe[1] is as follows: The disease began at the age of fifty-two years in the extremities of the lower limbs, and is said by the authors to have been the first case of the kind. The antero-external muscles of the legs first showed atrophy and the weakness progressed simultaneously with the atrophy. The poliomyelitis was strictly limited to the nerve cells of the anterior horns. The anterior roots and peripheral nerves were secondarily affected. No mention is made of the employment of the method of Marchi or of the condition of the tendon reflexes.

The case observed clinically by Dr. Moleen and studied from the pathological aspect by me may properly, I think, be regarded as one of chronic anterior poliomyelitis. The commencement in one lower limb, the paralysis preceding the atrophy, the comparatively rapid development of the symptoms, the absence of sensory disturbances, and the pathological findings justify this diagnosis. The prompt tendon reflexes are unusual, but it is probable that Bielschowsky's patient would have shown the same condition if these reflexes had been observed earlier in the disease. The patellar reflexes were exaggerated also in Lövegren's case. The degeneration of the crossed pyramidal tracts in our case was very slight and perceptible only by the Marchi method. When the Weigert method was employed no suspicion of degeneration of the pyramidal tracts could be entertained. The case, however, shows that the distinctions between amyotrophic lateral sclerosis and chronic anterior poliomyelitis are not always to be sharply made. The Marchi method does not seem to have been employed in the examinations of many of the cases of chronic anterior poliomyelitis reported.

It is noteworthy that pain was recorded as present in the cases of Nonne (Case I.) and Grunow, and that paræsthesia occurred in Oppenheim's second case.

MYOCLONUS MULTIPLEX: WITH REPORT OF A CASE.

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Historical. Since Friedreich in 1881 (Virchow's *Archiv*) reported for the first time his case of "paramyoclonus multiplex," some cases similar and many more dissimilar to it have found their way into the literature under the same appellation and caused endless con-

[1] Revue neurologique, 1902, p. 1075.

fusion to clinicians and students. Little comfort has been derived from the generic term myoclonia, with which it was intended to placate every neuromuscular spasm or condition in which muscular twitching was the only or most notable symptom. It was the fate of most of the essential myoclonias reported to the number of 100 or so, after much trimming and remodelling, to neatly fit into one or the other of the many accepted forms of neuroses (tics moteur) exploited principally by the French school of neurologists. Unverricht in a retrospect of all cases of myoclonus, 48 in number, collected by him up to 1891 commented on 6 as genuine paramyoclonus multiplex; 11 looked like it, 11 others were far removed, and the rest properly belonged with the tics.

One contemporary writer of eminence, Dana, saw fit in 1903 to revise the old extensive classification whose chief merit lay in the eponymic character of the nomenclature, and I append for purposes of comparison his differential grouping:

Myoclonias and their synonyms.	Dana's grouping of myoclonias.
I. Paramyoclonus multiplex, of Friedreich; astasic myoclonia, of Vanlair; multiple spinal myoclonus, of Lowenfeld; fibrillary chorea, of Morvan; fibrillary myoclonus, of Kny.	I. Myoclonia of Friedreich, or peripheral type, including myokymia.
II. Functional, or hysterical myoclonus multiplex, chorea major, chorea electrica, of Henoch.	II. Myoclonia of the functional or hysterical type.
III. Myospasmia; memory spasm, of Friedreich; habit chorea; chorea variable des degeneres; convulsive or spasmodic tic; tic general; Tourette's disease, myriadrit; palmus; tic neurosis, of Collins.	III. Myoclonia of the convulsive tic type, this last type being characterized by many associated spasms, choreic and tonic.
IV. Degenerative chorea, hereditary chorea. Huntington's chorea; myoclonus epilepsy; myoclonus type; myoclonia congenita of Seeligmuller (?); hereditary degenerative chorea of Sachs.	IV. Myoclonia of the degenerative chorea or "familial" or myoclonus epilepsy type, which is closely related with the third.
V. Infectious chorea, chorea minor, Sydenham's chorea; chorea electrica of Dubini (?); chorea electrica of Bergeron; senile chorea of Gowers (?).	V. Myoclonia, of the type of the infectious and symptomatic choreas.

It is, I believe, conceded that Friedreich's classic report of this rare disease in 1881 antedated all other descriptions, and yet within a few years the literature was enriched by the records of Löwenfeld, Remak, Seeligmüller, Francotte, and Kny, followed by the views of Bechterew, Marie, Unverricht, Lundborg, Wollenberg, Boettiger, Schultze, Moebius, Oppenheim, Tourette, Strumpell, Gowers, Russel, Dana, Hunt, Walton, Clark, and Prout.

At the outset it should be made clear that one series of cases, which Unverricht elaborately described and published under the name of "Myoclonia," differed widely from those of Friedreich. Unverricht dealt with familial types and reported cases of "associa-

tion-disease" (myoclonia with epilepsy) differentiating them from the independent disorder about to be cited. I say independent disorder with some hesitation, since Wollenberg is one of those who regards paramyoclonus as a feature in the symptom-complex of hysteria; Schultze places it with the forms of convulsive tic, to which view Dana also inclines; Unverricht classes it with the degenerative choreas, and Marie, Oppenheim, Bechterew, and Strumpell, for example, favor its recognition as a disease entity.

An epitome of the Friedreich case may the better serve to show how radical have been the departures from *the original* type and how by deviation and addition the literature has grown to its present proportions and provoked such keen controversy. Friedreich tells us of a combmaker, aged fifty years, who for some time had been a hospital patient suffering from pulmonary tuberculosis. The symptoms that brought him under Friedreich's observation developed after a violent fright, which he had experienced five years before at the sight of a bursting circular saw, which accident had inflicted neither personal injury nor bodily shock. The patient was a neuropath. Two weeks thereafter he developed sharp clonic rapid twitchings in a group of muscles in the upper and lower extremities. The twitchings amounted to spasmodic contractions at close intervals, limited mostly to the biceps and triceps of both arms and the forearm supinator longus. Of the leg muscles the vastus externus and internus and rectus femoris were elected to show these clonic spasms. In the face and over the back an individual muscle would occasionally contract. Only when the spasms were especially violent were the muscles thrown into locomotion; ordinarily synergetic muscular play was not observed. The spasms varied in frequency and intensity. The movements abated during sleep, but mere resting in bed had the effect of increasing them to even tetanizing strength. Work or voluntary movements brought the spasms under almost complete control. The dynamic motor power was not impaired—the electrical reactions remained normal; there was no idiomuscular swelling, so-called myoidema, upon stroke of the muscle volumen with the hammer. Cutaneous irritation, however, produced excessive muscular spasm. The patellar tendon reflexes were markedly exaggerated. The mental state was absolutely sound; pupils negative; all physical functions normal except for headaches and dizziness. Galvanization at frequent intervals led to a decrease in all symptoms and the patient was given his discharge shortly after.

Hunt[1] comments on the Friedreich case as follows: "The peculiar features of this affection—viz., the spontaneous contraction of individual muscles not singly under the control of the will, and the absence of the synergetic muscular action in the affected members,

[1] Journal of Nervous and Mental Disease, July, 1903.

were so at variance with the known characteristics of muscular contractions of cerebral origin as to render some other explanations probable. Friedreich, after a careful consideration of all the possible sources, as the brain, spinal cord, nerves, and muscles, proposed the hypothesis which referred the symptoms to an *excessive irritability of the anterior horn cells of the cord.* This theory was in harmony with the special clinical features of the case—*i. e.*, the single muscle contraction, the absence of synergetic action, the great exaggeration of the knee-jerks and their diminution as the motor symptoms subsided, the great susceptibility of the contractions to the peripheral stimuli and their cessation on voluntary innervation of the part."

PATHOGENESIS. More regrettable than the confusion which has arisen concerning the classification of this affection is the speculative attitude toward its origin.

Friedreich sought to locate the lesion for this unusual symptom-complex in the spinal cord, and although the weight of the evidence still favors this view, Hunt feels that pathological anatomy has thrown no positive light upon the subject. He infers this much from the negative results obtained after thoroughly conducted histological examinations in two cases, one investigated by Friedreich and the other by himself. In the original Friedreich case Schultze's examination of nervous structures and muscle tissue (the biceps) was entirely negative. Hunt's finding of a normal cord in his case of similar clinical type was equally disappointing, but he fared somewhat better in his study of the muscle fibres, which were found to be greatly hypertrophied and showing sarcolemma nuclei between the sarcous elements. In speaking of their presence Hunt says: "This is a peculiarity of the so-called red fibres of the lower vertebrates, a form of muscle which is more active and capable of more prolonged contraction than the white fibres, which constitute human muscle, although such red fibres are scattered in very small number throughout the muscular system in man." The thought that excessive and long-continued muscle twitchings and spasms could account for the muscle hypertrophy was dismissed when comparison was made with a slide of muscle tissue from a chronic progressive chorea of fifteen years' duration, in which the fibres were normal.

Neither in the protracted spasmodic conditions of cerebral origin nor in the class of muscular dystrophies and hypertrophies are such changes in muscle noted.

In an attempt to correlate Friedreich's theory of the possible spinal origin of the disease with his own observations of muscle change, Hunt concludes that a trophic relation may be responsible, thus strengthening the conviction in favor of cordal disease. That the anterior horn cells exercise a trophic influence resulting in atrophy of muscle is taken for granted when Hunt says, "it is

reasonable to assume the possibility of an increased trophic function with resulting hypertrophy."

The view of a cortical origin of paramyoclonus is more especially held by those who have been privileged to study the tissues of those rarer cases of paresis, chronic meningitis, and epilepsy associated with myospasms throughout their course. In an article entitled "Del Paramioclono Molteplice" Murri expressed the conviction, based upon 3 cases observed and autopsied by him, that disease in the Zona Rolandi, which he did not always succeed in finding, may and does give rise to polyclonus. There is something unsound in an argument that insists on the existence of a disease focus when all determinable evidence thereof is lacking.

Introductory to their paper on "The Nature and Pathology of Myoclonus Epilepsy," Clark and Prout say of essential myoclonus (paramyoclonus multiplex), "our knowledge of the pathological seat of essential myoclonus, while still defective, is tending more and more to place it in the cerebral cortex, where most other chronic convulsive disorders are being located. We have no doubt that even the milder types of essential myoclonus when studied in the light of more recent and definite histological changes of the cortex from cases of myoclonus epilepsy (the most marked and fatal form of the myoclonus) will disclose its pathogenesis to be in those cortical elements of the third or pyramidal layer and will show that the lesions belong to that form of cell degeneration which is so uniformly present in the second layer cells of the epileptic brain. If such findings prove true in a large number of cases of essential myoclonus the disease must be classed as an affection similar in pathogenesis and course with that of epilepsy; or, in other words, that there exists a soil of degenerative predisposition upon which is engrafted an autotoxic excitant."

Clark and Prout share with Unverricht in this somewhat positive assertion of a cortical origin of the disease and their deductions are made from the premises of myoclonus epilepsy. In reflection of the enormous number of cerebral cases in which small and numerous pathologic foci are found identical with those seen in myoclonus epilepsy, it is not far-fetched to ask why we do not see intravitam more of these twitchings and spasms of the paramyoclonus type. They are genuinely rare in the symptom complex of the many brain states whose underlying pathology is well understood. It may be safely contended that the myospasms associated with cerebral disease are not at all of the paramyoclonus type. They are neither so violent nor so rapid nor confined to individual muscles nor so widely distributed. They resemble the more coordinated movements of the average tic.

Experimental study has thrown little or no light on the subject. A muscle does not respond individually to an electrode be it applied ever so carefully to the motor cortex. The response is one of move-

ment of a group of muscles. Hunt states that Sano's experiments have a better hearing on this point and go far to demonstrate that the individual muscle is represented in the anterior horn cell.

Moebius regards paramyoclonus as an hysterical manifestation, and Dejerine, although less positive in that assertion, thinks it has much in common with familiar motor symptoms of hysterical nature.

In citing the following case I beg to remark on its clinical agreement with Hunt's understanding of myoclonus multiplex, "a term which should be reserved for that form of myospasm characterized by multiple spontaneous, isolated contractions of individual muscles."

A. W. P., a male, aged twenty-eight years, six feet tall, weighing 180 pounds, and by profession a prize fighter, was admitted to the neurologic service of the Cook County Institutions at Dunning January 1, 1905, and was there seen by me for the first time in April of same year.

Family History. Except for the fact that his father, who had always been a heavy smoker, died of a cancer of the lip at fifty-two, the patient's antecedents prove uninteresting. His mother is living and perfectly well at fifty-eight. Two brothers, one a pugilist the other a "buck dancer," enjoy good health, and of his two living sisters he has no knowledge.

Previous Illnesses. The patient states with much positiveness that prior to the onset of the present illness he had never known a sick day. Inquiry into his venereal history brings a confession of three attacks of gonorrhœa, but a local sore is stoutly denied and constitutional evidence of past syphilis is lacking.

His personal habits have been outrageously bad. As he says, "when not fighting men he was fighting booze." For fifteen years he has been a heavy drinker of hard liquor, and his sexual indulgence, following several years of rather pernicious masturbation, has been excessive. He has passed through all the vicissitudes that go with his chosen profession.

Present Illness. In the fall of 1902, in company with some fellow-fighters and trainers, he became as he calls it "paralyzed from drink." A party of four men consumed seven quarts of whiskey at one sitting. It was directly after this "drunk" that he felt and saw a twitching, "a jumping" of the abdominal muscles, which in the course of a few weeks had spread so as to have affected all the muscles of the trunk. Simultaneously fine muscular contractions appeared in the left hand and arm, rendering it too unsteady to be of any service. Careful inquiry fails to reveal the fact of a regular progression in these twitchings; they seemed universal, and in January, three months from date of onset, they had attained the dignity of spasms severe enough to make walking impossible.

Drink was necessary to steady his gait throughout the winter months of 1903. He had quit fighting altogether and devoted his

time to the training of other pugilists, punching the bag, drinking, and sporting. In the spring of 1903 he did odd jobs at a race-track, principally walking horses for exercise. During the summer he lived in out-door camps, and the following winter kept himself well enough under the influence of liquor to do snow-shovelling. The early part of 1905 was spent in Detroit, where he got sixty days for drunk and disorderly conduct.

At the time of commitment his spasms were so generalized and severe that he could not walk. Only by sitting down and shoving himself along could he move about. Throughout the entire illness there has never been a complete let up in the spasms. He states that occasionally days intervene when "the jumps" are milder and enable him to walk or stand, but the "hard spells," to use his phrase, may persist for days, weeks, or months without the slightest variation in intensity. At times he experiences hard, dull pain in the lumbar region and across the shoulders.

It would seem from what he says that the hardest spells are preceded by prodromata, described thus: A bright zig-zag light flashes before one eye, the right only, compelling him to lie down immediately; then follows a very intense frontal type headache, which only succumbs after indulgence in food or drink. His nerves then quiet down to a state of what he calls "ordinary spasms." He has never experienced symptoms that even remotely resemble an epileptic fit. There has never been loss of consciousness, frothing, biting of the tongue, bed-wetting, or tonic rigidity. Emotional excitement aggravates the spasms. Will power exercises no control.

Status Præsens (objective findings). The man presents a physique which attests to muscular development and power. The many batterings which his face and lips have received in the course of a fistic career and the flattening of the nose from repeated fractures lend a rather unlovely expression to his features.

It does not require the removal of clothing to note the lightning-like jerks that play through the various muscles of trunk and extremities, and every now and again become so severe as to give the impression of his being thrown from the chair to the floor. The muscle contractions impress one as being induced under the stimulus of an invisible electrode. So severe and shock-like are the seizures that locomotion is well-nigh impossible. Any effort at standing, even though supported, meets with difficulty from the violence of the spasms. The act of dressing and undressing is most tedious and cumbersome, since all attempted movements seem to aggravate to greater helplessness.

Resting in bed with all clothing removed, the generalized clonic seizures present a most unusual picture. In parts of a muscle one sees a faint quivering of a few fibrils. In a larger muscle, the fascicular type of twitching is evident; then again muscles singly or in groups spring forward in sharp tetanic contractions, while still

others are so clonically convulsed that locomotor effects result Here, there, everywhere, together or in rapid succession, the play of muscles goes on. During one extremely severe spell, which I witnessed, no muscles seemed spared; even the muscles of the eye, the tongue, and the occipitofrontalis were observed to share in the seizures. With the examining hands placed on the peroneal flexors of both legs at the same time the "tic" was distinctly appreciated on the left a second before it was felt in the right. Very close watch with the eye confirmed this succession. The tic appears bilaterally symmetrical, but is not bisynchronous. The entire left side is much more affected than the right, justifying the use of the term hemiparamyoclonus. Bernhardt has described a case of hemimyoklonia.

In the left leg muscles everywhere it was noted that a few minor tics both palpable and visible precede the major spasms, most of the latter being severe enough to throw the limb off the bed. On timing the larger seizures it was shown that they recurred about every three or four seconds and the slower minor twitchings occurred in the intervals about one second apart. Since the left-sided spasms are greater by far than those of the right, the motor effect is correspondingly greater and the left side the more helpless of the two.

The contractions everywhere were clonic, never tonic, and it would be impossible to declare any muscle or set of muscles free. The face, trunk, and periphery are alike the seat of constant spasms. The muscles chiefly involved are the deltoids, pectorales, flexors, and extensors of the legs and arms, the gluteals, abdominal, back, and thoracic muscles, as well as those of the diaphragm. Everywhere the rapidity and the intensity of the spasms varied. In the face their fibrillary character could be beautifully detected in the small muscles, the risorius and buccals. In the glutei a short wave could be distinctly seen and felt, and here the tic seemed slower, more rhythmical, reacting more like a muscle that shows a slight R. of D. to the faradic current; its bilateral character was more readily observed. In the brief absence of large clonic spasms, the fine fibrillary twitchings persisted even the small musculature of the feet and in isolated muscles that are not under control of the will. Hunt has observed this peculiarity in a case observed by him and commented on the isolated contraction of the supinator longus and sartorius muscles, which cannot be operated at will. Over the right pectoral the myospasm caused the muscle to form in a lump-like mass.

The speech of the patient is, from the combined force of a diaphragmatic and laryngeal spasm, too interrupted and explosive to be understandable. Before the patient could speak with a semblance of comfort he had to be given whiskey and chloral. With every severe seizure involving these muscles there is a loud inspiratory grunting sound as if the patient were choking. Deglutition is often a two-step or three-step act. Upon being given a slice of bread

to eat the spasm held a piece of it in the back of the throat, releasing it after a few seconds and so enabling him to complete the act of swallowing. The same sensation obtained in the act of drinking.

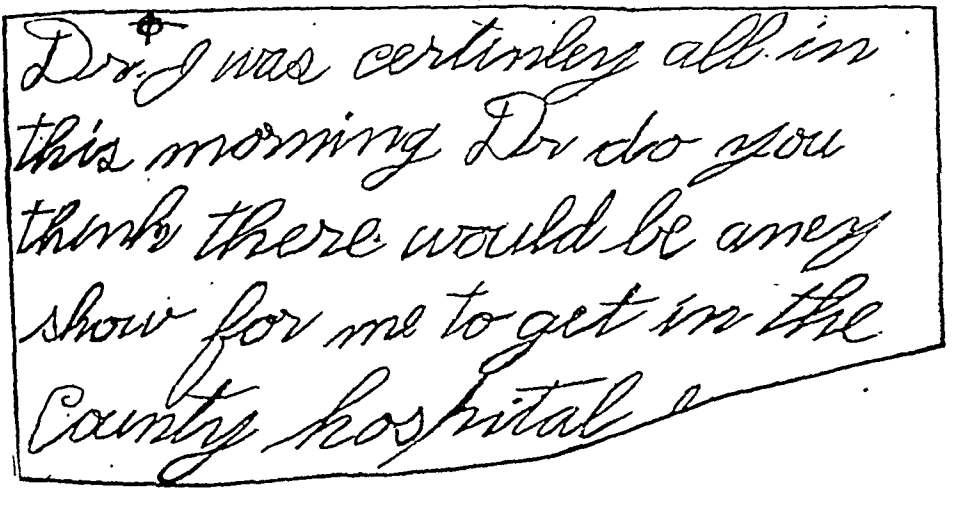
Bladder and rectum had never been incontinent nor disturbed, and the sexual power remains normal.

The muscular system is well developed, nowhere showing a pathological degree of atrophy or hypertrophy. Motor paralysis is absent and such disability as exists is entirely borne of the myospasms and the paresis or reduction of dynamic power can readily be attributed to disuse. The dynamometer registered 100 with the right hand and 70 with the left. *Myotatic* irritability is increased. Stroking the soles of the feet caused a notable increase in the twitching. An electrical examination was not made. The tendon reflexes everywhere are brisk, especially the jaw and knee-jerk. The Babinski toe sign was absent. The cremaster was well marked. The eyes are normal, the pupils react properly. The frontal eminences were unduly prominent. Sensation was nowhere disturbed.

The patient states that he feels better in the afternoon than in the morning. Lying on the left side is next to impossible, and keeping on his back is an equally great hardship; he gets only comparative comfort in any position, but favors lying on the right side. When first taken ill, he found that though bedridden by day, he could walk some at night, an anomaly which does not obtain now.

Despite this body and nerve-racking affliction the patient is uncomplaining, unemotional, and rather inclined to be witty. He has not suffered the least impairment of his mind.

I append an extract from a letter written by the patient while under control of whiskey and such irregularities in the tracing of letters as would be expected, nor does the up or down stroke show even so much as heavy shading from undue pressure in steadying the hand for the task. Free from the influence of whiskey writing is utterly impossible, as are all other acts of the will.



TREATMENT. During an all too brief period of observation several hypnotics and sedatives were tried and in nearly all instances with indifferent result. Sulphonal in 30-grain dosage given in hot milk and repeated in two hours had the effect of only slightly reducing the twitchings. Sodium bromide and the combined bromides in dosage of 1 to 2 drachms and upward failed to induce quiet, much less sleep. Hyoscine hydrobromate ($\frac{1}{60}$ gr.) hypo. had absolutely no effect on the spasms. Morphine sulph. ($\frac{1}{4}$ gr.) hypo. was equally inert. Choral in 10-grain doses by mouth had a quick sedative action where all other drugs failed, and 20 grains subdued the contractions for several hours. Better than all these in its sedative action was whiskey in 1-ounce doses. Given alone it would steady patient in about thirty minutes and make walking, writing, and eating an easy matter. Alcohol *added* to any of the drugs mentioned acted beneficially when the drug alone proved useless.

In Dana's case alcohol was without the least effect and I note what Clark and Prout say of various remedies used in their myoclonus epilepsy cases, "Alcoholic excesses usually excite a series of myoclonic contractions, yet small amounts of alcohol often markedly diminish the myoclonus for a time." Of chloral they say, "when given alone it has but little power in checking the myoclonic contractions and is practically of no value in diminishing the frequency or severity of the epileptic seizures."

A half-pint or pint of whiskey, which the patient on one occasion was allowed to consume in a half-hour or less, steadied every nerve and muscle in his body for almost an entire day. To sum up, then, whiskey or chloral or both proved to be the only successful agents at my disposal for allaying the myospasms. The diet and general hygiene were such as fall to the lot of patients in a large county institution. The patient was discharged unimproved on June 1, 1905, and immediately repaired to his home in Detroit.

I am indebted to Dr. Swan, senior physician at Dunning, for the use of some of his notes.

N. B.—The case was presented (in brief) at the May meeting of the Chicago Medical Society.

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INFECTIOUS ARTHRITIS: A BACTERIOLOGICAL CONTRIBUTION TO THE DIFFERENTIATION OF THE "RHEUMATIC" AFFECTIONS.

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I. Introduction.

It is surprising, considering the frequency with which "rheumatic" joints of unknown origin, and not referable to commonly accepted types, are met with in any general hospital or similar private practice, that so little is known of the actual etiology in this large group of cases.

The report here presented deals particularly with three cases of this nature in which the bacteriological findings have appeared of sufficient importance to warrant consideration in detail. The fourth case is one of typical acute articular rheumatism. In order to show their relationship, clinically and otherwise, with rheumatic disturbances in general, I wish to refer briefly to that classification of rheumatic joint affections which seems best to accord with our present knowledge.

CLASSIFICATION OF RHEUMATIC AFFECTIONS.

Exclusive of simple traumatic cases and of suppurative conditions of pyogenic or mixed bacterial origin, the term *rheumatic affections* is here used broadly to include cases in which joint symptoms form a more or less prominent feature.

1. Tuberculous and secondary syphilitic joints find a place here, as do also certain arthritides in which a specific bacterial cause has been determined—gonorrhœal, pneumococcic,* etc. Here, too, though with less certainty as to their exact nature, we may include joint involvements directly following specific infections elsewhere in the body[1] [2]—typhoid fever, scarlet fever, measles, dysentery (Shiga's), influenza.

2. As examples of arthropathies of nervous origin may be cited those of tabes, syringomyelia, neurasthenia, and of certain cases of peripheral neuritis.

* The majority of pneumococcic joints, however, proceed to suppuration. Cave, in Lancet, January 12, 1901, has reported thirty-one cases collected from the literature up to that time, nearly all suppurative.

3. The articular disturbance following administrations of diphtheria antitoxin illustrates cases of toxic origin.

4. Another class of cases may be attributed to hereditary and diathetic conditions—*e. g.*, those seen occasionally in hæmophilia. Gouty joints, both the acute and the so-called chronic types, have been variously classed as toxic or as diathetic.

This would account for practically all the sharply acute inflammatory conditions except those we are accustomed to designate as the acute articular rheumatism occurring in rheumatic fever. Reference will be made later to the claims of this condition to classification with the infectious arthritides.

5. Beside these there yet remain several other conditions which have been reserved to the last because of the great confusion and disagreement that has followed attempts to differentiate them; for this reason Osler and others have preferred to consider them collectively under the name of *arthritis deformans.* In this group it is probable, in the light of recent work, that we have to differentiate at least two distinct classes of cases—those in which *changes of an essential nature occur in the bones and cartilages* of the joints involved, and those in which *such changes are absent or are secondary* to the primary lesion. It seems possible that these two classes are also etiologically distinct, and that the latter are of infectious origin.

It is to this latter class that my first three cases belong. In referring to it I shall use the name first applied to it by Schüller[3]—*infectious "polyarthritis chronica villosa."* It will be more profitable to examine at once the character of these cases themselves before discussing the propriety of grouping them with others under the head of *arthritis deformans.*

INFECTIOUS POLYARTHRITIS CHRONICA VILLOSA.

Clinical Characteristics. This is an affection which may develop at any age, in infancy or in adult life. Marantic conditions predispose to and often definitely introduce it. It follows sometimes upon parturition. A varying degree of constitutional disturbance may mark the onset, though this is often entirely absent and the first symptoms may appear in the joint without any assignable cause. With little or no warning a joint may become sensitive and quickly exhibit signs of an acute inflammatory nature. The local picture at the onset is so similar to that in rheumatic fever that it needs no description. As in the latter condition, several joints are generally attacked in rapid succession, as a rule the larger articulations, though the small joints of the hand are frequently involved as well. The local symptoms are usually milder, but may approach in severity those of rheumatic fever. Some enlargement of the neighboring glands is not uncommon. A moderate leukocytosis is the rule in the early stage; the pulse rate is almost invariably high

(95 to 110) in adults, but there is generally little or no pyrexia. The heart is rarely affected; chorea and subcutaneous nodules* have not been noted.

So far there is little to distinguish these cases from mild forms of rheumatic fever; their *subsequent course*, however, is very different.

In the latter condition the course throughout is essentially acute, the *restitutio ad integrum* of the joints being rapid and characteristic. Directly the opposite is true of the class of cases in question. With frequently no interference with the general health the course is protracted, and the acutely inflamed joints of the early stage acquire a chronically congested and much hypertrophied synovial membrane which permanently deforms the joint. Recurrences of the acute condition after intervals of comparative quiescence are characteristic. From continued disuse shrinkage of the capsule may occur, and contractures of the surrounding muscles, eventually, though rarely, leading to true bony ankylosis, but the entire absence of essential lesions of the bone or cartilage sharply distinguishes it from the *atrophic* or *hypertrophic arthritis* of Goldthwait.[2] Chronic hydrops may continue to be a feature for a longer or shorter period; it is of the character of a transudate, never, apparently, exudative. When the process has not been unusually protracted so that contractures have occurred, the only interference with function is that presented by the hypertrophied synovial folds which act as foreign bodies in the joint. As in other conditions in which such villi are developed it frequently happens, however, that these become adherent to various portions of the synovial walls, and unless proper attention be given to breaking up such adhesions a permanent disability may result.

Pathological Characteristics. The examination of such joints after characteristic changes have developed shows a condition of villous hypertrophy of the synovial membrane. The character of the tissue varies considerably with the stage of the disease—in the recent cases frequently showing marked turgidity and congestion, later often acquiring a comparatively anæmic character. The histological picture varies accordingly. The occurrence of obliterating endarteritic changes may cause necrosis, with rupture of the pedicle and production of joint-mice. Occasionally a general synovial thickening takes place and definite villi are absent. After sufficient quiescence lipomatous transformation of villi is not uncommon; metaplasia into bone or cartilage is observed fairly frequently, and at other times deposits of amorphous lime salts. The fibrous capsule is generally thickened.

Very similar changes are produced in a variety of other conditions, due to local inflammations from various causes. Interest-

* Though mentioned by Bannatyne (Rheumatoid Arthritis, 1898, p. 71) it is uncertain as to what class of cases he there refers.

ing as these are, their consideration here is not to the point. Painter and Erving[4] have discussed the subject at some length, and with numerous references to the work of others. Schüller,[5] in 1900, in the course of a series of communications, described the histological differences in the synovial villi produced in various conditions, illustrating with cuts.

Compared with the *histological* picture presented by these other villi of various origins, it may be said of those arising in *infectious polyarthritis chronica villosa* that they differ from the former in showing a more extensive thickening of the synovial membrane and a tissue of more cellular character. Moreover, the character of the cells themselves differs with some constancy, for in addition to the large fixed cells with large, clear, vesicular nuclei which, together with their supporting framework of branching fibrous trabeculæ, form the structural background in almost all villi, we see here numerous plasma cells, and a disproportionate increase of infiltrating small round cells, and, in cases where the process is still active, great numbers often of polymorphonuclear leukocytes and eosinophiles. The lymphoid cells appear particularly as a continuous zone beneath the free surface, but nodular aggregations of considerable size are often met with in the deeper layers, especially about the numerous bloodvessels. The lymph channels are large and abundant. The free endothelial border of the synovial membrane often shows several layers of cells instead of the normal, single, flat layer.*

The histological picture is always beautiful. Though moderately characteristic in certain stages, it can never, of course, be relied on as in any way of diagnostic value. The gross anatomical picture is of more value, but it is the clinical features that are of chief importance. As to the value of the *x*-ray picture in the differential diagnosis from *atrophic arthritis* Goldthwait[2] speaks positively.

It is not my purpose to describe these cases further than will suffice to illustrate clearly the class with which we have to deal. It must be added that clinically they may display very considerable variation from the general type outlined above, *all characterized, however, by a course of a chronically infectious nature, polyarticular, with the production of permanent changes in the joints, affecting primarily only the periarticular structures, and having little or no tendency to display lesions elsewhere in the body.*

Relation to Arthritis Deformans. Nomenclature. It is necessary for the sake of clearness to touch briefly on this subject. Of the many terms applied to certain chronic deforming diseases of the joints, the most commonly used in this country is *arthritis deformans*, and it is just as well to disregard the numerous other terms synonymously employed.

* In one case (synovial villi removed from the wrist-joint in a case that had presented typical clinical and pathological features) the cells of the free border had acquired a cuboidal form.

Osler's classification is probably the best known. His subdivisions under this head are: (1) Cases with Heberden's nodes; (2) general progressive arthritis—polyarticular, and occurring both as acute and chronic forms; (3) monarticular forms; (4) vertebral —spondylitis deformans; (5) Still's disease.

Goldthwait, on the other hand, makes but three subdivisions, based rather on the pathology and supposed etiology. These are: (1) Hypertrophic arthritis*—including Osler's subdivisions (1), (3) and (4), characterized by essential hypertrophic changes in the bone, supposedly due to disordered metabolic conditions. (2) Atrophic arthritis, including cases of similar nature, characterized, however, by primary atrophy instead of hypertrophy, the changes first appearing in the articular cartilage and thence extending to the bone—it is evident that only part of Osler's subdivision (2) belongs here. (3) Infectious type, including the remainder of Osler's subdivision (2), together with all cases of Still's disease. The latter are included here in the belief that they are merely manifestations in childhood of the same conditions, Still having described only the most striking —*i. e.*, the fatal cases.

This last subdivision of Goldthwait's is identical with the class of cases I have called *infectious polyarthritis chronica villosa*. As these cases differ clinically, pathologically, and probably etiologically† from the hypertrophic and atrophic types there appears good reason to restrict the application of the term *arthritis deformans* to the latter two forms, which have important features in common. Goldthwait retained the "infectious type" under the general head, apparently as a concession to our usually accepted classification, to avoid confusion, and for lack of definite proof as to their etiology. Of more significance is his mention of this same class of cases in his article[2] entitled "Infectious Arthritis" (pp. 6 and 8).

Henceforth in speaking of *arthritis deformans* I shall have reference only to the cases of the hypertrophic and atrophic types.

The name of Max Schüller, of Berlin, deserves mention with this group of cases, for his is the honor of being the first to distinguish them as such. This he did first in a paper[6] presented to the XXI. Cong. der Deutsch. Gesellschaft f. Chir. in 1892.

Again,[3] in 1897, he differentiated them clearly from cases of *arthritis deformans* by the absence of essential changes in the articular structures proper, but was unwilling to commit himself as to their exact relation to ordinary acute articular rheumatism. His report was based on the observation of 56 cases. The condition

* In his earlier writings Goldthwait distinguishes *osteoarthritis* and *rheumatoid arthritis*. These terms are synonymous with *hypertrophic* and *atrophic arthritis*, which were subsequently adopted as being more accurate and less confusing.

† McCrae (Journal of the American Medical Association, October 8, 1904) states his belief that all three forms are of *infectious origin*, but there is no scientific evidence in support of this. As to the work of Bannatyne, which is supposed to favor this view, reference is made later.

he termed "*polyarthritis chronica villosa,*" and distinguished two types: (1) with chronic capsular thickening and great synovial hyperplasia, resulting in a greatly deformed joint with marked limitation of motion and persistent pain; (2) with capsular atrophy and villous formation limited to only a small area, pain a feature as above. As to his bacteriological findings I shall have occasion to speak later on. His descriptions are brief and clear, and are worthy of examination.

Subsequent observers, especially in England,* and certain writers in this country have referred to Schuller's work, but have failed to recognize the truth of his distinctions, and have confused his cases and their own with forms of true arthritis deformans. Thus, in reviewing the literature with reference to the special group of cases in question, it is always necessary to take into account the clinical and pathological features of the reported cases, and to disregard the various terms according to which their observers have classified them.

Speaking of the same group of cases distinguished by Schuller, Goldthwait[2] writes: "It is also possible, and indeed probable, in the light of recent reports, that organisms have been found, or will be found, which will explain some of the cases which, up to the present time, have been shown to be undoubtedly infectious, but in which the definite organism could not be found." These words may well serve as an introduction to the following report:

II. Report of Cases.

In the collection and disposition of the materials used for bacteriological purposes particular attention was paid to avoiding contaminations.

Chances of error. Technique.

This is mainly a consideration of the opportunities and possibilities of contamination. Where *aspiration* is employed there are three special sources of danger.

(*a*) Contamination from the *skin.* It must be conceded that this is possible, though reduced to a minimum by suitable preparation of the patient. In all my cases the parts were prepared as for a major operation, in most of them a bichloride poultice was worn for the preceding twenty-four hours. The organisms cultivated are not such as we recognize as the ordinary inhabitants of the skin.

(*b*) Contamination from the *air.* It is but fair to draw attention to this ever-present source of error. All manipulations were planned with reference to avoiding this. The syringe, after sterilization,

* The recent references by W. Hale White, and by A E Garrod to this group of cases are, however, definite Lancet, March 18, 1905

remained covered until required; it was handled with properly disinfected hands, and care was taken not to touch the needle itself. Inoculation of tubes was made at the bedside, plugs and mouths of tubes well flamed, and transfers carried out as rapidly as possible.

(*c*) Contamination from the *syringe*. This was avoided by using an instrument all parts of which could be boiled, being entirely of glass with the plunger wrapped with asbestos, and with a stiff rubber connection for the needle. These were boiled for fifteen minutes before using, and resterilized before aspirating a second joint of the same patient.

Cases in which material was obtained at *operation*. Contamination from the *air*. This is practically the only source of error here. My practice has been to have on the table, covered in the bag in which they were boiled, a graduated glass of suitable size and a small glass funnel. Before opening the capsule, and after bleeding is well controlled, the glass is taken from the bag and held close to the capsule, the wound being well retracted. The capsule is then nicked and contents allowed to escape into the glass.* The latter is at once covered with a fresh sterile towel that has not been exposed to the air, and as soon as possible the contents are rapidly transferred to a sterilized, cotton-plugged flask, the small funnel being used for this. If sufficient fluid is not obtained cultures are made directly from the joint on blood-serum tubes, and smears are likewise prepared. Any bits of tissue removed from the joint are at once folded between sterile sponges that have not been exposed to the air.

Notwithstanding all these precautions there remains, theoretically, a considerable chance for contamination. That this will seldom occur is shown: first, by the larger number of cases in which no organism was cultivated; secondly, by the consistent results obtained in the other cases where the same organism grew in pure culture in tubes inoculated from different joints of the same patient, or in tubes inoculated separately with the fluid, and with the tissue from the same joint. In the case of rabbits, where chance of contamination is much greater, it is worthy of note that out of thirty-nine aspirations from which cultures were attempted, all either remained sterile or yielded in pure culture the organism injected, excepting two tubes which were contaminated.

From 9 cases of various clinical types cultures from the joints were attempted on blood serum. Of these only 4 gave positive results. The negative cases were as follows:

Negative Cases.

No. 5. Mr. M. (Corey Hill Hospital, Dr. Goldthwait.) *Atrophic arthritis* (many joints). All cultures remained sterile. They were

* Dr. Leary has suggested a much simpler and safer method—viz., the aspiration of the fluid through the capsule after the skin incision is made.

taken from the knee-joint fluid obtained by aspiration, and later at operation, and numerous tubes were inoculated. The attempt was also made to grow cultures in large quantities of bouillon after Blaxall's* method. Three blood-serum tubes were also inoculated with bits of snipped-up synovial tissue from a small quantity removed at operation. No organisms were seen in smears fromthe fluid.

No. 6. Miss H. (Carney Hospital, Dr. Painter.) Considered to be an early *atrophic arthritis*. An afebrile polyarthritis with acute pain and slight effusion in left knee-joint. At time of aspiration the effusion had practically disappeared, but a few drops of clear fluid were withdrawn from the left knee-joint and two plain blood-serum tubes and one blood-smeared tube inoculated. The two plain tubes remained sterile, the third tube was doubtless contaminated. Smears from needle-point negative.

The culture medium in the third tube was blood serum that had been smeared with placental blood (collected aseptically and kept on ice in Ehrlenmeyer flasks). A number of such tubes were prepared and incubated for several days before use, but as many which were not used, but remained in the incubator, developed growths after a longer period (seven to ten days), no cases are reported as positive in which this medium was used.

No. 7. John W. (Carney Hospital, Dr. Painter.) *Infectious arthritis following influenza.* This was a comparatively recent elbow in which, however, synovial fringes had developed and proliferation had been stimulated in the head of the radius. Cultures were made from the synovial fluid, which, though thick and syrupy, was clear and but slightly in excess. Bits of synovial tissue, and bone removed from head of radius were also placed in separate tubes of the blood-smeared serum. No growths were obtained save such as were clearly contaminating. Smears from synovial fluid negative.

No. 8. Arthur L. (Carney Hospital, Dr. Minot.) *Acute articular rheumatism.* This was a first attack, in both wrists and knees and in one elbow, with mitral insufficiency. The arthritis subsided quickly and at time of aspiration only a drop or two was obtained from either knee-joint. Blood-serum tubes inoculated, but remained sterile; smears from needle-point negative.

No. 9. Miss A. (N. E. Baptist Hospital, Dr. Goldthwait.) *Chronic infectious arthritis* (many joints). This represents the border-line cases, in which, though of evident infectious origin, and without essential bone or cartilage involvement, permanent peri-articular change has been produced, without, however, well-marked villous proliferation. It seems likely that subsequent attacks might produce the typical picture described by Schuller.

* *Lancet, April 25, 1896* It was realized, however, that the cases with which Blaxall worked, though described as "rheumatoid arthritis," were undoubtedly not of the atrophic type, but of the variety differentiated by Schuller.

The left knee-joint was aspirated under cocaine anæsthesia, with rigid aseptic precautions, and three or four drops of fluid obtained. Two tubes were inoculated, one of plain and one of blood-smeared blood serum. Smears from needle-point negative. Two days later a pure culture was discovered on the plain tube—a motile, pale-staining bacillus, decolorized by Gram's. The organism grew in discrete, round, elevated, colorless colonies, not visible to the naked eye until the third day; when fused, indistinct granular patches appeared. It was very difficult to obtain smears, apparently from unusual coherence of the organisms, and the firm, membranous-like character of the colonies, so that it was necessary to pick a colony up bodily and crush it on the slide; even then few isolated organisms were found compared with those that remained *en masse*. Sub-cultures on fresh blood serum developed slowly, the colonies later acquiring a yellowish hue.

On the second day after aspiration the blood-smeared tube also showed a growth similar in character to that described, but because of inability to obtain the organism in smears the colonies were regarded as perhaps oil-droplets or something else extraneous, unlikely as this appeared. A subculture on fresh serum was attempted, however, and yielded a pure culture from which smears were successfully made, showing presence of the same organism above described from the original—not the parent—tube. This organism was not of the character of the contaminating cocci that grew on many of the blood-smeared tubes. Moreover, it was found in pure culture on *both* the original tubes, one of which contained plain blood-serum. Nevertheless it is thought unfair to include it with the report of positive cases, although of sufficient interest to warrant consideration because of its similarity in certain respects to the spore-bearing organisms isolated in three of the positive cases. Besides it bears, so far as studied, a considerable resemblance to Schüller's and Blaxall's bacilli. It is hoped that another opportunity may be offered to study this case.

Positive Cases.

Case I.—Frank A. L., Massachusetts; aged twenty-eight years; machinist. Admitted April 27, 1904. Carney Hospital, orthopedic service, Dr. Painter.

Complaint. Stiffness of various joints; persistent swelling in knees.

Family History. No tuberculosis, neoplasm, or joint affections.

Personal History. Gonorrhœa at age of eighteen; no complications. Measles at age of twenty-four. No history of chronic diseases (except of joints), chorea, or any nervous disorders. General health always good. No history of traumata or surgical operations.

Joint History. At age of seventeen, eleven years ago, developed moderate pain, with stiffness, in both hips; onset sudden, preceded

by chill, following exposure to cold and wet; not in bed, but off feet for two to three weeks; no subsequent attack in hips. For past four years has noted moderate tenderness about shoulders, with some stiffness in those joints. One year ago, April, 1903, having gone to bed in excellent health, woke with severe dull ache in both knees; considerable swelling, but no redness or heat. Condition continued unchanged for four months, not preventing work; since then knees have remained swollen, but painless, excepting on attempting to use joints after some inactivity. Eight months ago noted stiffness, without pain, developing in neck. Appetite good; sleeps well; bowels regular.

Physical Examination. Poorly developed, emaciated, pale. *Chest* symmetrical; expansion (across nipples) limited to 1.5 cm. *Lungs* negative. *Heart* of normal size and position; both sounds clear; at the apex a soft systolic murmur, faintly transmitted to the axilla, not heard in back. *Abdomen* negative, spleen and liver not felt. *Glands:* moderate, soft enlargement of posterior cervical, right axillary, and both inguinal groups. *Reflexes:* knee jerks present and equal; ankle clonus fairly distinct on left; no Babinski.

Special Examination. Spine: Stands with considerable list to the right; back shows a long, gentle kyphotic curve, of postural variety, with a double scoliotic curve, and no apparent rotation; cervical spine slightly flexed forward; practically no motion whatever permitted by spine, except to a slight degree in cervical region. *Shoulders:* rotation free; all other motions much restricted. *Elbows, wrists, hands,* negative. *Hips:* habitual slight flexion and abduction hips very stiff, but with some manipulation, flexion, abduction, and adduction can be carried to almost normal limits; rotation about one-half normal; symmetrical atrophy of both thighs. *Knees:* both are symmetrically enlarged throughout the synovial outlines of the joint; no redness or heat; not sensitive to palpation; patellæ freely movable and do not float; swelling feels boggy and soft, irregular masses can be felt to glide under the examining fingers; no bony thickening made out; both knees can be completely extended; flexion beyond 135° in right and 100° in left, is painful and resisted by patient. *Feet* and *ankles,* negative. *Temperature,* 101°. *Urine,* negative. *Blood:* not examined.

Operation, April 29, 1904; Dr. Painter. Incisions made on both sides of patella of left knee about 10 cm. in length. The fibrous capsule was somewhat thickened, the synovial capsule much thickened and very villous. The villi were quite vascular, and in greatest abundance about the border of the patella and along the edge of the femoral condyles. There was no excess of fluid in the joint, but a blood-serum tube was inoculated with a loopful. The cartilage was clear and glistening, and not eroded. The ligamentum mucosum was considerably thickened. The villi here and about the condyles and patella were trimmed out and the capsule closed

with interrupted silk. Skin closed with continuous subcutaneous silkworm gut. A voluminous dry dressing was applied to the extended knee.

The skin sutures were removed at the end of one week, and moderate passive and active motion begun. On the twelfth day functional use was permitted. Operation on the other knee refused by patient.

May 14, 1904. Has been walking about for past two days with no pain or apparent weakness. General condition excellent. Discharged, with flannel bandage about knee, to report in one week.

The highest temperature was on admission, 101°, varying thereafter from normal to 99°.

Nothing has been heard from the patient since last note, and attempts to locate him have failed.

Bacteriological Report. A loopful of serum, obtained from the left knee-joint at operation, was inoculated into a tube of plain blood serum. Upon this, in pure culture, developed an organism whose morphological and cultural characteristics are described later in the outline. This tube was put on ice, and for lack of opportunity was not studied until five months later. It was feared the culture had died out through drying down of the medium, but being a spore-bearer it was readily revived in pure culture, and showed the same characteristics as before. Its identity is further established by the finding in the tissue removed at operation of an organism of the same morphology and staining (Gram's) properties, as well as by the results of animal experimentation.

The synovial tissue removed at operation was hardened in 80 per cent. alcohol for histological examination. It consisted of a large handful of coarse, thickly-set villi, not very vascular. Sections stained by the Gram-Weigert method showed the presence of the bacillus in small numbers, lying mainly in the lymph channels or cells at some distance from the free border. The character of the tissue corresponds histologically with the description in the introductory section. The animal experiments are described under a later heading.

Case II.—Aaron B. R., Nova Scotia; aged thirty-eight years; marine engineer. Admitted January 5, 1905. Carney Hospital, orthopedic service, Dr. Painter.

Complaint. Pain and swelling of right knee.

Family History. Negative; no joint affections.

Personal History. Has had various diseases of childhood. Gonorrhœa three times, the last fifteen years ago. No history of chronic diseases, nor of any previous joint troubles.

Present Illness. Seven months ago began to have sharp pains in right knee, radiating up the thigh, aggravated by frequent kneeling at work. One month ago joint was subjected to a sudden

strain; since then pain has been continuous, and joint swollen except for brief periods of enforced rest. Kept at work until a few weeks ago.

Physical Examination. Well developed and nourished, color good. Lungs, heart, and abdomen negative. Condition of glands not recorded. All joints negative except right knee. *Right knee:* Capsule much distended and patella floats. Thickened irregular masses can be felt, apparently within the capsule. Motions of joint considerably restricted. Circumference across patella 5.5 cm. greater than on left side; 3 cm. atrophy of right thigh and calf. *Temperature* normal. *Urine* negative. *Blood,* hæmoglobin, 80 per cent.; red blood corpuscles, 6,000,000; white blood corpuscles, 12,600.

Operation. January 10, 1905. Dr. Painter: Incision made on inner side of patella of right knee, about 8 cm. in length. Before opening the capsule all bleeding was controlled and wound well retracted. Through a small incision in the capsule about three ounces of serous fluid was allowed to escape into a sterile flask. Capsular incision was then extended. Bone and cartilage were normal. The whole synovial membrane was thickened and rough. Villous masses depended from the internal alar ligament, and a pannus-like fold of synovial membrane overlay the inner surface of the internal condyle. These tissues were removed, and operation completed as described in Case I., the after-treatment being carried out in same manner.

January 24th. Discharged, wearing flannel bandage over knee. Temperature practically normal throughout stay in hospital.

Bacteriological Report. 1. *Serum.* About ℥iij were obtained, rather syrupy and cloudy but not flakey, of light urinous color. 2 or 3 c.c. were removed with a sterile pipette, transferred to a tube of plain blood serum, and incubated at 37° C. No growth ever appeared on this tube, which was discarded after two weeks.

Two centrifuge tubes, which had previously been plugged with cotton and sterilized one and a half hours by dry heat at 150° C., were half-filled with serum removed with the sterile pipette, and contents centrifugalized for three minutes. From the bottom of one of these tubes two or three drops were removed with a freshly sterilized pipette, inoculated into a tube of plain blood serum, and incubated at 37° C. Smears were also made from the bottom of the centrifuge tube. These, like smears from the uncentrifugalized serum, showed only a quantity of fibrin and small numbers of red and white blood cells, probably blood flowing from nick in the capsule. The inoculated tube showed no visible growth after twenty-four hours; the surface of the serum was bathed with the water of condensation. Twenty-four hours later a plentiful growth of discrete, colorless, pinpoint colonies appeared in pure culture over the whole surface.

The remainder of the serum, about ℥iss, was transferred to a sterile Florence flask and incubated at 37° C. Four days later a drop from this flask showed presence in small numbers of an organism, morphologically the same as cultivated from centrifugalized portion, but clumped and immotile.

2. *Tissue.* This consisted of five or six small bits of rather lipomatous nature and not very vascular. The most vascular-looking bit was snipped several times with a pair of sterile scissors, quickly transferred with sterile forceps to a tube of plain blood serum, and incubated at 37° C. After twenty-four hours no growth was evident, and surface was bathed; after forty-eight hours it showed a growth identical with that from the centrifugalized serum, and examination showed presence of the same organism in pure culture. The cultural and morphological characteristics are later described in outline.

The remainder of the tissue was placed in 10 per cent. formalin for histological examination. Cut sections showed no peculiarities other than described in the introduction. The organism isolated does not stain by Gram's, but sections stained with polychrome methylene blue and eosin show the presence of the bacillus in very small numbers in the tissue.

Animal experiments are recorded later.

CASE III.—Annie M. B., Massachusetts; aged forty years; single; music teacher. Admitted December 17, 1904. N. E. Baptist Hospital, Dr. Goldthwait.

Complaint. Pain and swelling in many joints, especially hands and elbows.

Family History. Mother died of cancer of stomach. Father died fourteen years ago of typhoid fever. Definite "rheumatic" history on the part of two maternal aunts, both being on crutches and crippled in many joints for ten years and twenty-five years, respectively.

Personal History. Measles and mumps in infancy. Typhoid fever fourteen years ago, at same time with father. No history of traumata or early joint troubles. Never robust, but always in fair health until attack of typhoid fever.

Present Illness. Began at age of twenty-six years, seven weeks after full convalescence from a typhoid fever unaccompanied by complications. An acute swelling developed suddenly in both wrists, coming and going irregularly. In the next few years various other joints became involved, especially of hands, elbows, knees, and feet, with occasional pain referred to hips. At various times the swellings have been very considerable, hot, red, and painful. They were never aspirated. Twelve years ago hands were so troublesome that work (stenography) was impossible. Much improved a year later, and for five years grew steadily better. An attack of influenza (?)

four years ago seemed not to affect joint conditions. Eighteen months ago had erysipelas of face, after which no trouble until a year later, when the frontal sinus was opened on account of a progressive swelling in left orbital region; character of pus not determined. Since that time, however, all joints have been worse, particularly in the hands, and the trouble has since appeared in the shoulders. The general health at present is fair, but the patient is in a nervous condition.

Physical Examination. Fairly well developed, poorly nourished, pale. *Heart* and *lungs* negative. *Abdomen* negative; spleen and liver not palpable. *Glands* slightly enlarged in neck and in both axillæ; epitrochlear glands not palpable.

Special Examination. Both sternoclavicular joints tender; slight swelling about both maxillary joints, and movements painful; spine apparently not involved. *Shoulders:* the right allows normal motion, but with much crepitation; the left shows same condition, but motions are all guarded. *Elbows:* both show normal amount of flexion, but stop short 20° of complete extension. *Wrists* and *hands:* distinct swelling of practically all the metacarpophalangeal and several interphalangeal articulations, with slight amount of ulnar deflection of both hands. Hips apparently not involved. *Knees:* motions somewhat guarded and painful in both joints; outlines practically normal. Both feet and ankles negative.

x-Ray: A good negative of both hands and wrists shows practically no alteration from normal in either bone or cartilage.

December 22, 1904. The two most swollen joints of the hands—the right middle metacarpophalangeal and the left middle proximal interphalangeal—were aspirated for bacteriological purposes.

23*d and* 25*th.* 6 c.c. and 8 c.c. of Aaronson's antistreptococcic serum injected into thigh muscles.

28*th.* Has been having evening temperature of 100° to 100.5°, with morning remissions to normal; pulse, 100; respirations, 24. To-day temperature rose to 101.4°; pulse, 120; respirations, 26. There was pain in right nipple line over liver. Transferred to Dr. Badger, account of pulmonary conditions.

30*th.* The picture is clinically of pneumonia, but examination is negative; some few friction rubs over base of right lung in front.

Leukocytes, 24,000. Urine negative. Respiration short, painful, shallow. No sputum.

January 1, 1905. Bronchial respiration over left base, behind.

3*d.* Pneumonia fully established in left lower lobe. Heart free.

6*th.* A to-and-fro pericardial rub without signs of fluid.

25*th.* Three ounces clear fluid withdrawn from pleural cavity. No culture made.

February 4*th.* Delayed resolution slowly clearing. Heart conditions have cleared. General condition much better. *Joint symptoms have improved in a remarkable way.*

March 11th. Lung is not wholly cleared; a little bronchial respiration still heard at left base, otherwise resonance and respiration good throughout. There has never been any sputum. The highest temperature was 103.2° on January 1st.

Discharged by Dr. Badger, well of pulmonary conditions* and with joints in better condition than for months.

Bacteriological Report. As noted above, one joint of each hand was aspirated under aseptic precautions, several days before onset of pulmonary symptoms. From each joint one small drop of serum was obtained, and from each one plain blood-serum tube was inoculated, and incubated at 37° C. Unfortunately no smears were made from the needle points. At the end of two weeks one tube was discarded, being sterile.

The tube inoculated from the right middle metacarpophalangeal joint showed no visible growth at end of twenty-four hours; surface was bathed with water of condensation. Twenty-four hours later a fine growth had appeared over surface of medium—colorless, glistening, pinpoint colonies in pure culture. Subcultures were made on various media. The organism is described in the outline later.

Animal inoculations are also recorded later.

The following case differs from the preceding three in being one of typical acute articular rheumatism.

CASE IV.—Solomon K., Russian Hebrew; aged nineteen years; papermaker. Admitted January 9, 1905. Carney Hospital, medical service, Dr. Minot.

Complaint. Knees painful.

Family History. Not obtained.

Personal History. "Rheumatism" twice before, last attack one year ago. Denies all acute infectious and venereal diseases.

Present Illness. Began December 14, 1904, with pain in ankles and left elbow. Three days later admitted to Boston City Hospital with temperature of 102.5°, pulse 120. Throat was found normal; no glandular enlargements. The left elbow was tender but not red; the right ankle and foot were tender, red, and swollen. Was given antirheumatic treatment, and being somewhat improved was discharged, January 5, 1905, to United States immigration authorities. January 9th admitted to Carney Hospital, saying he had caught cold the day before and could not walk because of pain in knees.

* Bannatyne,[7] speaking of the arthritic type to which this case belongs, says (p. 27): "I find that about 3 per cent. of my cases developed either pneumonia or pleurisy during the time they were under my care, and about 17 per cent. developed endocardial or pericardial conditions after the onset of the disease."

Morphological and Cultural Characteristics.

	Bacillus No. 1. Case 1.	Bacillus No. 2. Case 2.	Bacillus No. 3. Case 3.	Bacillus No 4. Case 1.
Morphology: shape	Long plump rods, with slightly rounded ends.	In first cultures diplococcoid, but after transplantation — plump rods of moderate length, with slightly rounded ends.	Pleomorphic.[2] Lanceolate diplococcoid forms; on favorable media single and diplobacilli, with rounded ends, generally lying in parallel clumps.	Long, plump rods, with slightly rounded ends.
arrangement	Single, in pairs or in long chains: no true branching forms found.	In pairs chiefly; also single forms and short chains, no true branching forms found.		Single, in pairs, or in long chains; no true branching forms found.
size	Width 0 5–0 63 μ; length 1–4.9 μ; averaging 1–2 μ.	Width 0 6 μ, length 1–2.4 μ.	Width 0 6–0 8 μ; length of elements 0.7–1 μ; length of pairs 1.08–2 36 μ.	Width 0.72–0.76 μ; length 1.6–1 μ; averaging 1 5–2.5 μ.
capsule	None demonstrated.	None demonstrated.	None demonstrated.	None demonstrated.
spores	Clostridium type, readily produced in old cultures.	Clostridium type; considerable numbers in 3 day blood-serum cultures.	None demonstrated.	Clostridium type; great numbers in 3 day bouillon cultures.
Motility	Actively motile.	Actively motile.	Non-motile.	Actively motile.
Staining properties	Stains readily with aniline dyes, and by Gram's.	Stains readily with aniline dyes, decolorized by Gram's; occasional polar granulation in old cultures	Stains readily with aniline dyes, and by Gram's.	Stains readily with aniline dyes, decolorized by Gram's.
Cultural chars	Facultative anaerobe.	Facultative anaerobe.	Aerobic, practically no anaerobic growth.	Facultative anaerobe.
blood serum, 37° C.	Grows vigorously, in 18 hours covering surface of medium and water of condensation with a thick, tough, corrugated pellicle of pale rose color; liquefaction of medium fairly rapid; not checked by icing.	Delicate growth as raised, viscid, colorless thin line; discrete colonies are round, raised, flat, colorless, pin-point, increasing to pin-head size in 2–3 days; no further increase.	Individual colonies same as of bacillus No 2, but growth even less marked along streak, and not progressive.	Very vigorous growth, liquefaction beginning in 12 hours, and producing deep, viscid, yellowish furrows; a heavy, smooth, white pellicle rapidly covers water of condensation; discrete colonies minute, round, raised, moist, whitish, translucent, quickly fusing.
plain agar, 37° C.	Streak: grows well, in 24 hours showing a broad, elevated, white streak with moist, wrinkled surface, increasing but little after 48 hours, becoming powdery; individual colonies are minute, colorless dew-points, rapidly fusing. Stab: fair continuous growth, not increasing.	Streak: same as on blood serum at 24 hours, colonies later becoming ivory white. Stab: fair filamentous, continuous growth by third day, not increasing.	Streak. only a few smooth, round, flat, slightly elevated, discrete colonies, colorless, of pinpoint size, slowly increasing to diameter of 2 mm. and becoming whitish and crenated. Stab: very faint growth in depths.	Streak: after 24 hours, a slightly elevated, ivory white, broad streak, with irregular, smooth border; heavy, smooth, white pellicle over water of condensation; after 18 hours little change; no liquefaction. Stab: moderate, continuous, white growth after 24 hours, soon producing small, tuberous offshoots.

potato, 37° C.	At 24 hours a good growth; several large, viscid, white globules along an ill-defined, faint, pinkish streak, suggesting appearance of watermelon fibre; at 48 hours globules are collapsed, a wide-spread, rose colored growth in dew-point globules; later develops a blood-red color, not diffused.	Grows with difficulty, producing no visible changes.	Limited growth; moist, raised, whitish, almost invisible; typical forms in smears from scrapings.	At 24 hours raised, moist, sticky, lemon-colored growth of considerable vigor, in broad, irregular streak; medium slightly darkened; by fourth day growth has increased and become whitish, potato discolored brown; no further advance.
alkaline bouillon, 37° C.	Quickly becomes turbid; small waxy globules float on surface, later coalescing to form a firm pellicle, like honeycomb, over the whole surface.	Grows well, producing general turbidity in 24 hours.	Grows fairly well, producing slight general turbidity, and small amount of white, granular sediment.	Marked, rapid turbidity; by third day waxy scales float on surface, later uniting to form a dry, friable white pellicle.
litmus milk, 37° C.	Pellicle formed as on bouillon, finally firm enough to resist on inverting tube; slight alkaline reaction by fourth day, followed by bleaching; gradually acidified after 10 to 14 days; no definite clot.	Grows well, a faint alkaline reaction developing slowly.	Grows well, a faint alkaline reaction developing slowly.	A firmer pellicle than on bouillon; alkaline reaction on second day, followed by bleaching; turns acid in 7 to 10 days, with late formation of clot, digested slowly.
glucose gelatine, 20° C.	Stab: infundibuliform liquefaction, gradually reducing surface to level; fair growth in depth, no gas; pellicle faint rose in old cultures.	Stab: slowly produces slight, brown, granular growth in upper half, not advancing; no gas; no liquefaction.	Stab: barely perceptible whitish growth throughout stab, not progressing; no gas; no liquefaction.	Stab: infundibuliform liquefaction, gradually reducing surface to level; thick, sticky pellicle; no gas.
His' semisolid, 20° C.	No gas.	No gas.	No gas.	No gas.
Dunham's peptone, 37° C.	No indol.	No indol.	No indol.	No indol.
at 20° C.	Grows well on blood serum, but without pigment, and less vigorously than at 37° C.	Grows fairly well on blood serum, but soon dies out.	Grows weakly on blood serum.	On blood serum same as at 37° C., but more slowly.

[1] Fresh twenty-four-hour bouillon cultures stained with Loeffler's methylene blue.

[2] The unusual forms are described under section on "Animal Experimentation."

Physical Examination. Well developed and nourished. Pharynx congested. *Lungs* negative. *Heart* impulse diffuse over large part of præcordia; apex in fifth space, 11 cm. from midsternal line, not enlarged; sounds regular; blowing systolic murmur over whole præcordial area, loudest at apex, transmitted to axilla and back; second pulmonic sound accentuated; diastolic murmur, heard loudest at base in pulmonic area. *Abdomen* negative; liver and spleen not felt. *Knees*: Right shows but little swelling, not red or hot, moderately sensitive to pressure. Left somewhat swollen and warmer than right, but less sensitive. Both patellæ free, but motions are guarded. Aside from the knees the second phalangeal articulations of the middle fingers in both hands and the right wrist-joint were swollen.

Temperature on admission 100.5°. *Pulse*, 90. *Urine*, negative. *Blood:* Hæmoglobin, 90 per cent.; red blood corpuscles, 5,200,000; white blood corpuscles, 11,600.

The treatment is not of interest in this connection. By January 22d the joint conditions were considerably improved, but still complained of pain in elbow. The temperature, never higher than 100.5°, came down slowly. To escape the immigration officers the patient suddenly disappeared from view, January 29th.

Bacteriological Report. January 12th. Both knee-joints were aspirated under ethyl-chloride anæsthesia, after scrubbing with 80 per cent. alcohol. They had previously been prepared as described under the heading "Technique." From the right knee about two drops of serum were obtained and inoculated into a tube of plain blood serum; unfortunately not enough was left to inoculate a second tube. Smears were made from the needle point. The same description applies to the left knee. The smears were stained with gentian violet, and with Loeffler's methylene blue; a prolonged search revealed nothing but large mononuclear leukocytes in small numbers.

The tubes showed no visible change after incubation for eighteen hours; the surfaces of media were bathed with the water of condensation. Forty-eight hours after inoculation* pure cultures of the same organism had developed in both tubes, described subsequently in the outline.

Animal inoculations were carried out and are recorded in a later section.

CHARACTERS OF THE ORGANISMS FOUND IN THE FOUR POSITIVE CASES.

These are described in considerable completeness, not because the organisms themselves are of special interest, but for the better comparison with others they may resemble and with others found

* This forty eight hour incubation period does not suggest contamination, it was later observed in all cases in which organisms were recovered from joints of inoculated animals

in cases of the same clinical types. It has not been possible to identify any of them with previously described organisms; in the attempt to do so reliance was placed chiefly in Chester's Manual.* (See tables pages 1066 and 1067).

ANIMAL EXPERIMENTATION.

Value of Evidence Afforded by Animal Experimentation. Three facts of importance here are to be deduced from the work done in this direction:

1. The well-established difference that various animals display, (*inter se* and compared with man), in their reactions to many organisms.

2. The greater resistance in general shown by animals as compared with man, more particularly in the case of organisms of slight pathogenicity.

3. The ability of a variety of *entirely distinct organisms* to produce in the joints of animals *changes indistinguishable clinically or pathologically.* This is well illustrated by the comparison of results obtained by Shaw[8] and by Cole,[9] in their milder cases, with those in my own series. Shaw worked with an organism from acute rheumatic joints. Cole employed virulent strains of streptococcus pyogenes.

From this it follows that:

1. The exact reproduction in animals of the lesions in human joints is, *a priori,* not to be expected.

2. Failure to produce *any* well-marked joint lesions is not to be interpreted as evidence of particular significance.

3. Positive results are no more than suggestive.

Report of Experiments.

In this work rabbits were inoculated, some intravenously and some directly into the joint. For the latter purpose the knee-joint was used; the left side was selected for experimentation, the right (with one exception, No. II., Case 1) was reserved for control. Both joints were shaved for comparison, and frequent observations were made both of local and general conditions. It may be said here that in no case did the general condition appear influenced by the treatment. Moreover, the animals inoculated intravenously never showed the slightest signs of articular disturbance, although large doses were administered in three cases, and in the fourth (No. IV., Case 1) 1.5 c.c. was injected, and the organism used had been passed several times through joints in which it excited an acute process.

* A Manual of Determinative Bacteriology, Macmillan Co., 1901.

Case 1. Rabbit I. Left knee.

October 5th. Received an initial dose of ɱviij bouillon suspension of a fresh blood-serum culture.* Only a transient inflammatory reaction followed, but after six weeks there gradually developed a periarticular thickening with signs of fluid in joint. At end of two months this condition was well established. Nine days after inoculation a pure culture was recovered by aspiration.

December 16th. Reinoculated with ɱij bouillon suspension from forty-eight-hour blood serum; the organism used was from a pure culture recovered from joint of Rabbit II. at autopsy.

The chronic condition became decidedly more marked, and joint was much distended with fluid; there was a persistent limp.

January 19th. Thirty-four days after last injection animal was chloroformed. *Left knee:* Capsule considerably distended and thickened; typical villous proliferation of synovial membrane; no acute inflammatory signs; bone apparently normal. Capsule contained about 2 c.c. of tenacious, whitish fluid containing a few flakes, which smears showed to be of nature of mild exudate; no organisms found in smears. Pure culture of original organism cultivated from joint fluid. The right knee appeared normal, as did all the internal organs. Joints were preserved as specimens.

Rabbit II. Both knees.

October 7th. Right knee inoculated with ɱviij from a pure blood-serum culture from joint of Rabbit I. (aspirated twenty-four hours after first injection).

10th. Left knee received ɱx suspension from recent blood serum culture. Four days later a pure culture was recovered and smears showed presence of a mild exudate, a few bacilli appearing in pairs. No clinical signs appeared in either joint.

27th. Died as result of an old process in lungs. Externally both joints appeared normal. The *right* showed only slight villous changes; smears and culture negative. The *left* contained an excess of fluid of exudative character, as shown in smears; there was distinct synovial thickening with congestion and, in places, typical villi. A pure culture was recovered from this joint. In paraffin sections from the synovial membrane histological changes were found, differing in no essential detail from those in the original human joint. Sections stained by the Gram-Weigert method showed the presence of organisms similarly situated in the tissues and identical in appearance with those found in the human villi. The liver showed small foci of coccidial disease. Both lungs contained large abscesses; the left was converted into a single large cavity containing about ℥ss of thin white pus. The nature of the latter process was not determined, but it clearly had nothing to do with the joint conditions and was evidently the cause of death.

* In the following notes, unless otherwise stated, it is to be understood that suspensions of twenty four hour blood serum were used for inoculations.

Rabbit III. Left knee.

October 12th. A pure culture from the joint of Rabbit II. was used; ɱviij were injected. A slight, transient, inflammatory reaction followed; a slight limp developed later.

December 16th. Reinoculated with ɱij from forty-eight-hour blood-serum subculture of organism isolated at autopsy from left knee of Rabbit II. Result slight.

24th. A final inoculation with ɱviij suspension from forty-eight-hour blood-serum culture, taken from joint of Rabbit I., resulted in a gradually increasing swelling, without acute signs, but causing limp. Eighteen days after inoculation a pure culture was recovered from the joint; smears showed existence of a considerable exudate.

The clinical signs had all disappeared at end of four weeks, but some capsular thickening persisted.

March 21st. Three months after last injection, chloroformed. *Left knee* showed merely a moderate degree of villous proliferation; smears and cultures negative. Joint saved as specimen. The right knee and internal organs appeared normal, and cultures from the heart blood, spleen, liver, and kidney were sterile.

Rabbit IV. Vein of ear.

January 18th. A twenty-four-hour bouillon subculture from joint of Rabbit III. was used; ɱxv were injected intravenously. No effects observed.

24th. 1.5 c.c. of a twenty-four-hour bouillon from same source injected. No effects whatever. Animal was still well until April 5th, when used for other purposes.

Case 2. Rabbit I. Vein of ear.

January 24th. 2 c.c. of twenty-four-hour bouillon culture, intravenously; no effects.

February 1st. Subcultures were made on blood serum from organism recovered from joint of Rabbit II. The growth on four twenty-four-hour blood-serum tubes was suspended in 5 c.c. of bouillon and injected into the vein. No effects. The animal is still well after eleven weeks.

Rabbit II. Left knee.

January 24th. ɱiv of twenty-four-hour bouillon caused only mild inflammatory signs, gone in forty-eight hours. The only time the organism was recovered from the joint was four days after this injection; it grew in pure culture; a very few organisms were also discovered in the smear from the joint fluid.

30th. Injected ɱiv suspension from forty-eight-hour blood-serum culture recovered from same joint. A slight reaction with moderate swelling, gone in two days.

February 16th. ɱv injected.

26th. ɱx injected.

In all, four inoculations were made but a satisfactory reaction was never obtained.

March 14th. Seven weeks after first inoculation, animal chloroformed. *Left knee* appeared practically normal, save for a few pale villi below the patella. Paraffin sections were made of these and the tissue was found lipomatous, in this respect resembling parts of the human tissue in this case. In one place a deposit of lime salts was found in the capsule. Staining with polychrome methylene blue and eosin failed to show presence of any organisms. There was no picture of acute infiltration, as in most of the sections from the human joint. Smears and cultures from this joint were negative. The right knee was normal, also the internal organs, cultures from which remained sterile.

Case 3. Guinea pig. Subcutaneous injection.

Of a twenty-four-hour bouillon culture $\frac{3}{4}$ c.c. was injected beneath skin over abdomen. Three weeks later a soft enlargement of glands in both groins appeared. No constitutional disturbance could be made out, and after three months animal was alive and well.

Rabbit A. Left knee.

January 19th. ɱiij of twenty-four-hour bouillon provoked little reaction.

28th. ɱvij from twenty-four-hour blood serum caused considerable swelling and some limp; practically normal by the fourth day.

February 8th. ɱx caused considerable swelling and some limp. Aspiration after five days showed presence of exudate and active phagocytosis in joint; culture sterile.

20th. Only a periarticular thickening remains. ɱiij injected. (Any effects of this inoculation were obscured by the development of an abscess located near, but outside the joint, having apparently nothing to do with the injections. Similar abscesses were present beneath the skin in other places before the injections were begun; the condition is very common in rabbits. The abscess in question disappeared in about ten days). A polar-staining bacillus, in pure culture, was obtained from the extra-articular abscess. As it was decolorized by Gram's it cannot be identified with the Gram-staining organism found later in sections from the synovial tissue.

March 2d. ɱx injected. No acute signs, but a considerable capsular distention and limp resulted, persisting until animal was killed.

The organisms were never recovered from the joint, though several attempts were made. As early as forty-eight hours after injection all organisms appeared to have been included by the phagocytes.

20th. Two months after first inoculation, animal chloroformed. The right knee and the internal organs appeared normal; cultures

from the latter remained sterile. *Left knee* contained about mvij of tenacious grayish exudate, from which cultures were sterile. Fibrous capsule normal, but there was a considerable uniform thickening of synovial capsule. A few minute villi hung by thread-like pedicles from the ligamentum mucosum; a few also projected from above the patella. These latter were sectioned in paraffin and stained by the Gram-Weigert method. Histologically, sections showed a moderate synovial thickening with presence, in considerable numbers, of the new tissue-forming cells; there was only a moderate degree of infiltration with small round cells and polymorphonuclears. Occasional sharply stained bacilli were found singly and in pairs. The latter were identical in size and appearance with certain forms occurring in the cultures from this case.

The following experiments were made with the view of identifying, if possible, these organisms with the ones injected—pleomorphism on artificial media having already been observed in the latter.

Rabbit IV., Case 1, was used, and on two different occasions 0.5 c.c. and 1 c.c. from twenty-four-hour cultures on blood serum were injected into the left knee-joint. Aspiration after twenty-four hours and eighteen hours, respectively, showed practically all the organisms included in phagocytes and incapable of staining by Gram's; cultures remained sterile. 25 c.c. of cloudy forty-eight-hour bouillon was then injected into the peritoneum, and after six hours a small quantity was withdrawn. In smears only a few organisms, and these of short forms, were found; many phagocytes appeared but the disappearance of the organisms seemed mainly due to bacteriolysis. Typical pure cultures grew in discrete colonies on blood serum.

Rabbit B. of this case was then similarly injected with 50 c.c. intraperitoneally. Two and three-quarter hours later a small quantity was withdrawn. Smears showed same picture as above, short pairs staining by Gram's being found free and in the leukocytes. Typical pure cultures grew in discrete colonies on blood serum.

After a few days both rabbits showed little sign of infection; it is too early to make any further report.

From the pure cultures in the last two cases smears were made on second and third days, and stained by Gram's. The majority of organisms were short diplobacilli; small coccus forms also occurred, and a small number of single long rods. The latter were measured with the micrometer and compared with the similarly stained organisms found in the tissue from Rabbit A. Here are the comparisons:

From cultures on blood serum: length in four cases: 1.9μ, 2.4μ, 3.1μ, 3.7μ.

From stained sections of synovial membrane: length in two cases: one diplobacillus, elements 1.3μ, pairs 2.8μ; one single rod, 3 (3.0μ). One or two longer forms in the tissues could not be measured, not lying in a horizontal plane.

Without cultures from the tissue the proof is incomplete, but the failure to obtain any growth from fluid aspirated on six different occasions speaks for the singleness of the infection.

Rabbit B. Vein of ear.

January 28th. A sterile water suspension of growth on four twenty-four-hour tubes of blood serum, intravenously, produced no noticeable effects. Ten weeks later this animal was used for intraperitoneal inoculation as noted above.

Case 4. Rabbit I. Left knee.

January 19th. ɱv of twenty-four-hour bouillon caused a fair inflammatory reaction with limp, lasting six days.

28th. ɱvij of twenty-four-hour bouillon caused less acute inflammatory change but more swelling and limp.

February 5th. ɱviij suspension of pure culture recovered from joint, and grown forty-eight hours on blood serum, produced a good local reaction with limp, and persistent swelling.

16*th.* ɱij as above from a recently recovered pure culture. No acute reaction.

28*th.* Swelling about gone. ɱxij caused a transient acute reaction, followed by a progressive swelling and limp, clearing after ten days.

March 18th. Eighteen days after last inoculation, animal chloroformed. The right knee and internal organs were normal. Cultures taken from the liver and spleen showed a few colonies of cocci, not pyogenic, and clearly not related to the experimental infection. *Left knee* contained slight serous exudate, from which the bacillus, in pure culture, was grown, and smears from which showed rare organisms, free and in the leukocytes. The synovial membrane was not congested, but bore patches of fine villi, a portion of which were taken for examination. A curious body, of the size, shape, and appearance of a small cantaloupe seed, lay in lower end of the lateral pouch outside the external condyle. It was attached by a long, thread-like pedicle to the inner side of the opposite condyle, where the synovial membrane had a fibrinous coating. Histologically, it was found to be organizing exudate. The villous specimen sectioned in paraffin, and stained with polychrome methylene blue and eosin, presented a beautiful histological picture, comparable to that in Case 1, but no organisms could be found after long search through many preparations.

Rabbit II. Vein of ear.

January 28th. 4 c.c. of twenty-four-hour bouillon.

February 6th. 3.5 c.c. suspension containing twenty-four-hour product of four tubes inoculated with pure culture from joint of Rabbit I.

After ten weeks no effects whatever have been detected.

III. Interpretation of Results.

This necessitates a consideration of the reports of other workers in this field. The findings in cases of acute articular rheumatism are not included here.

Bacteriological Reports of Other Observers.

1. *Schüller.* This evidence is considered first, as it is the most extensive and applies without question to the described class of cases.

His first report[6] was in 1892, when he mentioned finding constantly, in cut sections of tissue from the joints, the same organisms described in later papers.

1893.[10] By tapping joints (with cannula, allowing serum to drop directly on media in tubes and plates) he obtained the same bacilli found previously in histological sections. They are described as *short, plump rods, showing polar granulation* distinct from spores; the latter appear, however, in old cultures. Rods measure 0.75μ to 0.99μ x 2.24μ to 2.76μ, and stain by ordinary methods, though easily decolorized; no note here or elsewhere of Gram's method. Grew well at 25° C. in dark. In gelatin stabs, small white granules along course of puncture, going on to liquefaction; plates show whitish-gray points. On agar, similar plaques; the same on potato. Cultures ten to twelve months old were revived and proved infectious. Inoculation into rabbits' joints of 0.5 to 1 gm. caused marked swelling of permanent character and persistent limp. Animals were killed at end of two months, prior to which original organisms were recovered from inoculated joints. Pus was never found in the experimental joints; synovial membrane bore, in places, pale and brownish villous fringes, sections of which were shown to contain the organism injected. The uninoculated joints displayed no changes. Subcutaneous injections caused no suppuration, only slight local induration, but doses of 0.5 to 1 gm., it is said, were fatal in twenty-four hours. Injection of like quantity into joint produced nothing like this fatal picture.

1896.[11] A quick method is described for demonstrating the organisms in human tissues. At operation, when the fringes protrude through the wound, they are washed off with sterile water or $HgCl_2$, split open with a fresh knife and a sterile glass slide drawn across the newly cut surface. Smear is air-dried and stained with carbol fuchsin.

1897.[3] In sixteen arthrotomies, in cases of the type differentiated, in smears from the freshly cut villi (after method described above), and later in sections from these tissues, the bacillus was constantly demonstrated, generally easily, as the organisms are actually in the tissues and much less frequently free in the joint fluid. In aspirating the needle should be thrust, he says, not into the joint cavity but into the synovial tissues, where the organisms actually are. Of twenty-three aspirations, six were negative. In the pathological tissues the organisms are found mostly at the site of the greatest changes. Cocci often appear together with the bacilli, both in aspirated material and that obtained at operation; in one aspiration cocci only were obtained. These appear identical with the pyogenic cocci and are not regarded as playing any important role, certainly not a causative one. A comparison is made with cases of joint tuberculosis, where the tubercle bacillus is often found in small numbers, while pyogenic cocci are abundant. The present bacillus is not easily demonstrated in sections, because so readily decolorized.

A later paper,[5] in 1900, adds nothing bacteriologically.

2. *Bannatyne,*[7] *Wohlmann and Blaxall,*[12] 1896. Next to Schuller's this is the most complete bacteriological report bearing on our subject. It is therefore necessary to examine carefully the nature of the cases from which the evidence is deduced.

It is clearly emphasized (p. 79[7]) that cases of the hypertrophic type of arthritis deformans are to be excluded, and that it is from certain acute polyarticular cases only that the organisms were isolated. These cases are defined in pp. 91 and 92, from which the following is quoted: "Dr. Still, in Clifford Allbutt's *Medicine,* says there is in children a disease which presents important differences both in its clinical symptoms and morbid anatomy, as well as one which is clinically analogous to the rheumatoid arthritis of adults. The first type of the disease is characterized by chronic progressive enlargement of the joints . . . the joint thickening affects the tissues round the joints, rather than the bones or cartilages. . . . The pure rheumatoid arthritis in children he considers to be characterized by bony thickening and lipping about the joints. . . . Now, his description of the first class corresponds with my description of what we see in adults in the acute form of the disease, and also his second with the chronic form."

It is from adult joints of the first type, acute at the onset but developing permanent changes not involving bone or cartilage, that the following bacteriological evidence was obtained. Further descriptions (pp. 93 and 94[7]) bear out the conviction that Bannatyne is here dealing, in part at least, with the *polyarthritis chronica villosa* of Schuller, with whose work, and that of Chauffard and Ramond,[13] he compares his own bacteriological results.

To turn to these now, as summed up in the conclusion of Blaxall's report:[12]

"1. In the synovial fluid in eighteen cases of rheumatoid arthritis an organism has been demonstrated, which is constant in its characteristics.

"2. The organism is a minute bacillus, exhibiting marked polar staining. It is difficult to stain and easily decolorized.

"3. The organism can be grown in culture media, and presents striking characteristics. In beef-broth it gives the appearance of gold dust; and on agar-agar and serum its growth is almost invisible.

"It does not grow on nutrient gelatin at ordinary temperature.

"4. It is present in the blood of severe cases.

"5. It has not been found in the synovial fluid from distended joints due to other causes."

Two years later Bannatyne[7] states that in the few sections he was able to obtain, the same bacillus was found in the cartilages, synovial membrane, and periarticular tissues.

A special method for cultivation and an elaborate technique for staining were employed. The organism is described as averaging 0.6μ x 2μ, not occurring in chains, non-mobile, decolorized by Gram's, and in general requiring special care in staining. For further accounts of cultural characteristics, etc., reference should be made to Blaxall's report.[12]

Inoculations of mice, guinea-pigs, and rabbits were without results.

The bacillus is differentiated from Schüller's. It is mentioned in comparison with that found by Chauffard and Ramond, which, it is incorrectly stated (p. 83[7]), they "cultivated" from the synovial fluid.

Though a firm believer in the infectious origin of these cases, Bannatyne is uncertain as to the role played by the organism he has found.

3. *Von Dungern and Schneider,*[14] 1898. This is an account of a single case, very like, except for its fatal result, to my Case 1.

Man, aged forty-four years; acute polyarticular attack, subsiding after five months, leaving heart lesion; complete freedom for twelve years, when a second attack left him with permanent joint changes and some spinal rigidity; these conditions were still present five years later, when death occurred from inanition. At autopsy, characteristic changes were found in the joint synovial membranes, but with thinning of cartilage.*

The bacteriological report is of interest. It is based on a study of: 1. Aspirated fluid from finger-joints and blood from vein of arm (taken six months before death). 2. (Taken eighteen hours after

* On the basis of this latter fact the authors thought to distinguish this case from the type described by Schüller. It has already been pointed out, however, that such changes are not uncommon as secondary to long disuse or unusual severity of the local process, both of which features were present in this case; the patient had been completely bedridden for seven months before death.

death), fluid from several joints, heart blood, spleen, liver, kidneys, mucus from gall-bladder. Nothing was obtained from blood, spleen, or kidneys.

In smears a small diplococcus was found from the gall-bladder and cultivated on various media. The same organism was cultivated from the joint fluid and the liver. Morphologically, it much resembled diplococcus lanceolatus, grew in pairs, singly, or in chains up to ten, the pairs being 0.6μ wide x 1 to 2μ long, involution forms occurring. On agar small, discrete, transparent colonies were produced; on potatoes a white, granular growth, not elevated; on glucose agar after twenty-four hours a heavy confluent growth; milk acidified and coagulated in twenty-four to forty-eight hours; viability on artificial media demonstrated for at least three months. It was not identified with any previously described organisms. The joint lesions were reproduced by intra-articular inoculation in rabbits; importance was attached to the destructive changes* produced in the cartilages (not caused by Schuller's organism nor by control injections of pyogenic and other cocci, although villous and purulent conditions resulted from the latter). The diplococci were recovered and cultivated four to six weeks after injection.

From the liver and gall-bladder of this case, beside the diplococci, one other organism was found, from its morphology, cultural characteristics, and pathogenicity for animals, probably bacillus coli. From the joint fluid taken intra vitam a non-pathogenic coccus also grew; from that obtained postmortem there occurred, together with the diplococcus, a non-pathogenic bacillus.

The following conclusions were reached:

Not much importance is attached to these other organisms, yet it cannot be proven that the last two did not take part in the joint processes. Lesions once produced in joints render the latter favorable seats of infection. A good illustration is our finding there organisms of ordinarily such slight pathogenicity. It is possible that the diplococcus infection itself was not established by one invasion, but by successive attacks, these organisms being particularly abundant in the chronically diseased gall-bladder.

4. *Chauffard and Ramond*,[13] 1896. In a communication entitled "Des Adénopathies dans le Rheumatisme Chronique Infectieux" descriptions are given of seven cases. Excepting two that are probably of gonorrhœal origin these are evidently of Schuller's type. In all cases the anatomically related glands were distinctly enlarged; in two, valvular heart lesions existed. The bacteriological evidence is admittedly slender, and was sought in but one case. The joint fluid was twice aspirated and inoculated directly into tubes and animals, with negative results. The two

* The little reliance to be placed in inferences drawn from lesions produced in animal joints has already been referred to

epitrochlear glands were extirpated with strict asepsis. Of each a part was triturated and inoculated into culture tubes and various animals, with negative results; in histological examination of the remaining parts no organisms were found. Smears previously made from cut surfaces of the glands, and from the joint fluid, showed apparently the same organisms—short, fine diplobacilli, staining with the ordinary dyes, and not decolorized by Gram's.

Because of the frequent reference made to it by various authors, this report has been detailed to show its admitted lack of bacteriological value. It will not be referred to again.

5. *Bouchard and Charrin*,[15] 1891, 1894. These reports are also frequently referred to. The finding of pyogenic cocci in twenty-two cases of chronic joints is related, but the bare statements to this effect carry with them no conviction and do not require our consideration.

DEDUCTIONS.

It is not justifiable to base an opinion as to the etiology of a large group of cases, however much they may resemble each other clinically, upon a study of so few of its members. So much incredulity has been shown by the profession in general, with regard to the bacteriological reports previously made in rheumatic cases of various types, that pains have been taken in this paper to go into much detail.

Infectious Polyarthritis Chronica Villosa.

Case 1. The chain of evidence, so far as it is to be considered as such, is complete. The organism was isolated in pure culture from one of several joints in which the process had been recently active, and clinically typical. Histologically, it was found alone in the synovial tissue, included in the cells in an evident causal relationship. It was cultivated on artificial media. Injected into the joints of rabbits, it there provoked both acute and chronic processes, in nowise differing clinically or pathologically from those in the human joint. The organism was isolated repeatedly in pure culture from the animal joints, and sections from the tissues of the latter showed the presence in small numbers of this organism alone, similarly situated to those in the human tissue. Subcutaneously and intravenously moderate doses of exalted virulence were without effect.

Case 2. The organism in this case was isolated in pure culture, both from the synovial fluid and from the tissue, and grown artificially. Its presence was demonstrated in the human tissue, as in the preceding case. Though recovered in pure culture from the animal joint into which it had been inoculated, it failed there to produce any marked changes. Large doses intravenously were without effect.

Case 3. From one of two joints which were aspirated an organism was obtained in pure culture, under conditions that left little doubt that it played some role in the morbid process. Animal experimentation produced the same satisfactory results as in Case 1. Large doses intravenously proved harmless.

From each of these three cases a single different organism was isolated, apparently capable of considerable resistance under artificial conditions.

The findings in these cases differ from those of Schuller, who found in his own cases the same organism constantly. Bannatyne likewise, in the same type of cases, constantly found a single organism, but it was not Schuller's. It is impossible to explain the constancy of Schuller's results or of Bannatyne's. It is clear that they worked with the same type of cases as my own.

From the reports of previous observers, then, we should be inclined to believe that these cases are undoubtedly of bacterial origin, but that a variety of organisms may be responsible for the mischief. My observations serve to confirm this belief.

Nevertheless, even with a chain of evidence as complete as furnished in my Case 1, the possibility must not be overlooked that we may have been dealing throughout with organisms which invaded the joints only after the working of some other process had rendered them liable to infection. There can be no doubt that this may occur. In Case 2 the traumatic factor seems to have determined the unusual localization in a single joint; it is conceivable that in the absence of this factor the organism found there would have come to an untimely end without producing any more effects than in the healthy rabbit into which it was inoculated.

But the evident infectious character of the onset and of the whole clinical picture in these cases is suggestive. In the same way that predisposition toward all disease in general is determined, the local infectious process appears to be secondary to some factor which lowers the general or local resistance, rather than to any other preceding involvement of the joint—in other words, the joint disturbances are doubtless, *ab initio*, simple infections, but like all conditions of this nature, are much more frequent in individuals whose general condition is below par. As to the constitutional disturbances that frequently mark the onset, we may conceive of them in two ways—as manifestations of a general infection that soon becomes localized in the joints, or, as seems less likely, as caused by secondary general intoxication or actual infection succeeding an initial joint infection.

As to the probable *portals of entry* of such organisms, the same considerations apply here as in the case of a great variety of ordinarily saprophytic or weakly pathogenic organisms, which, under favorable circumstances, are capable of provoking local or even general disturbance. It is not within the province of this report

to deal with this phase of the subject. I would merely refer, in passing, to the important work of Adami[16] in this direction, and its abundant confirmation by subsequent observers. A recent communication by Wrzosek[17] promises to shed new light on this matter.

Acute Articular Rheumatism.

Case 4. In this case the same organism was isolated in pure culture from each knee by aspiration. It was artificially cultivated and reproduced satisfactory lesions, clinically and pathologically, in the animal joint into which it was inoculated. It was several times isolated from the latter in pure culture, but could not be demonstrated in the lesions.

With regard to the causal relationship of this organism to the local joint disturbances, the same conclusions apply as in Case 3. The actual heart lesions in acute articular rheumatism, apart from the joints, are too various to consider here.

Relationship of Acute Articular Rheumatism with Infectious Polyarthritis Chronica Villosa.

To the great number of observations recorded in the bacteriological study of acute articular rheumatism I can only refer in general. The results are more at variance than in the less investigated field of infectious polyarthritis chronica villosa. The sum total of evidence, however, is in favor of a varied bacterial origin. Even the emphatic claims of recent English observers to have found a specific cause in the so-called "micrococcus rheumaticus" have been well answered.[9]

Clinically and pathologically, as I have been at pains to point out, the class of cases described by Schüller are distinct from acute articular rheumatism.

Commonly coming under observation only after the chronic condition has developed, their infectious nature is often not recognized, nor is the futility of commonly applied methods of treatment—(dietary, hydrotherapeutic, massage, etc.)—appreciated. The frequent indications for operative interference* explain the interest of these cases to the orthopedic surgeon.

Bacteriologically, however, there is no evidence to show that there is any essential difference between acute articular rheumatism and infectious polyarthritis chronica villosa. The difference in the subsequent course of the two conditions is apparently due to differences in the infecting organisms and in the degree of disturbance they are capable of provoking.

This conception of a similar though not identical etiological

* It is to be noted that in resorting to surgical measures care must be taken to exclude *atrophic arthritis*, as such treatment is to be applied to this type only in selected cases.

relationship is no indication, as I have said, that the two types should not be sharply separated clinically. That this differentiation cannot always be complete must be very evident; points of resemblance occur when, in the chronic type, the general disturbance is considerable and the heart is affected, as in Case 1.

The necessity of a similar bacteriological study of a large number of cases is clear.

It is surprising that with the great interest in bacteriological research in this country, we should have failed to recognize the significance of Schüller's and of Bannatyne's work, and that we have contributed so little to the investigation of this large and important group of cases. No satisfactory work has been done in the bacteriological examination of blood in these cases. This should be a field of special interest, for it is to be pointed out that practically all the results reported in bacteriological study of acute articular rheumatism proceeded from the investigation of materials* other than the joint fluid.

To Dr. Timothy Leary, of Tufts College Medical School, in whose laboratory this work was carried out, I am grateful for much assistance and friendly courtesy. The kindness of Dr. Charles F. Painter, in providing me with the clinical material, is gladly acknowledged. My thanks are due also to Dr. J. E. Goldthwait, to Dr. J. J. Minot, and to Dr. George Badger for clinical notes and materials.

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* From a fairly complete list of the most important bacteriological work in acute articular rheumatism I have made out a table showing the character of the findings. Out of the positive results of 91 examinations from different materials, 18 were from the blood, 9 from joint fluid, and the rest from various other sources (autopsy materials, excised tonsils, urine, etc.). This does not show the proportion of cases in which the joint fluid was examined without results; there it would appear very much smaller.

A FATAL CASE OF STOKES-ADAMS DISEASE WITH AUTOPSY, SHOWING INVOLVEMENT OF THE AURICULOVENTRICULAR BUNDLE OF HIS.

A PRELIMINARY COMMUNICATION.

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The symptom-group known as Stokes-Adams' disease was first recognized by Adams in 1827, but more accurately described by Stokes in 1846. In recent years the subject has been discussed by Huchard in France, His, Hoffmann, and Jacquet in Germany, and by Prentis, Edes, Osler, and Babcock in America.

The symptoms that characterize the affection are: 1. *Slow pulse*, the rate falling temporarily or permanently to 30, 20, or even less. 2. *Cerebral attacks*, such as vertigo, syncopal or epileptiform seizures, unconsciousness. 3. *Pulsation of the veins in the neck* exceeding in rate the pulsation of the arteries twofold, threefold, fourfold or more. The disease was first recognized in old persons and in such as presented clinical or postmortem evidences of cardiac and vascular disease. Later instances were met with in younger persons and in some cases no lesion of any sort was found at autopsy. In Edes' series reference was made to the condition of the heart and vessels in 31 of the 35 cases that came to autopsy. In 26 of these sclerotic and myocardial changes were observed. In some cases lesions of the nervous system, such as compression of the medulla or changes in the vagus and cardiac plexus, have been described.

Recently, the resemblance of the clinical symptoms to the phenomena of "*heart block*," as produced by physiologists, has been pointed out. Gaskell first showed that constriction of the circular layer of muscle at the auriculoventricular junction of the heart of tortoises causes a cessation of the rhythmic action of the heart so that the auricles and ventricles become independent in their contractions, the former beating more rapidly than the latter. Various physiologists have confirmed this discovery and have discussed the nature of the mechanism which controls the rhythmic contractions. The prevailing opinion of physiologists at the present time is that the impulse to contraction originates at the large venous openings into the auricles and is propagated downward through the auricle into the ventricle within the muscle tissue itself. In a review of the evidence bearing on the transmission through the

muscle of the impulse to contraction, His[1] states that there are grave difficulties in the way of those who believe that nerve centres are the basis of cardiac activity. Rhythmic contractions certainly occur in the severed apex of a frog's heart after the contractions have been set in motion by some excitation. "Automatism," or *power of automatic contraction,* is therefore wanting, but there is present a *capacity for rhythmic contractility* after a stimulus has been given. Automatism in the frog's heart is found only in parts adjoining the auriculoventricular zone and the sinus region where there are ganglion cells. It does, however, also exist in embryonal hearts before ganglion cells are developed, though the experiments of Fano showed that even in such embryonal hearts the automatism is greatest near the sinuses and decreases progressively toward the apex of the ventricle. Physiologists agree that the ganglion cells at the venous sinuses are the principal source of the impulses for cardiac contraction, but Gaskell and Wooldridge proved that the nervous connections between auricles and ventricles may be severed without cessation of the ventricular contraction, and Krehl and Romberg have shown that in the adult heart the ganglia may be wholly separated without influencing cardiac contraction. The effect of the operation in the experiments of the latter authors was so slight that the heart maintained a blood pressure of practically normal degree. It would seem, therefore, that the impulse for contraction is transmitted from the sinus region to the auricle and ventricle through the muscle cells rather than through any nervous path. As a further evidence suggestive of this view Gaskell found that by stimulating the aortic bulb contractions could be made to run from ventricle to auricle; that is, in a direction the reverse of the normal. These experiments His regards as strongly antagonistic to the assumption that the impulse flows through a nervous mechanism.

If then the impulse travels through the muscle cells, it is necessary that a muscular connection be shown to exist between auricle and ventricle. Wm. His, Jr., first accurately described this connection in the mammalian heart in the form of a bundle of muscular tissue extending from the right side of the interauricular septum to the interventricular septum immediately below the *pars membranacea.* The "auriculoventricular bundle of His" is approximately 18 mm. long, 2.5 mm. broad, 1.5 mm. thick (Retzer). His' observation has been confirmed by Retzer, Bräunig, Humblet, and others. Though this bundle is an insignificant structure, physiological and anatomical evidence points to it as the pathway of the stimulus to contraction. In his experiments with Graufner, His found that by introducing a delicate knife through the auricle and dividing

[1] Wien. med. Blätter, November, 1891.

the auriculoventricular bundle in the septum (in rabbits) the auricles and ventricles ceased beating at the same rate. When the experiment was accurately performed the resulting condition was that of complete heart block, the auricles and ventricles beating independently of each other. A similar result has been obtained by Tigerstedt, who succeeded with a special appliance in compressing the auriculoventricular connections without hemorrhage and in producing the condition of heart block.

The most convincing experiments in this direction are those of Erlanger, who operated on the intact heart of dogs and other animals. With the aid of a specially devised curved needle, acting as a clamp, he was able to reach the auriculoventricular bundle of His by introducing the needle behind the posterior aortic leaflet and pushing the point through the interventricular septum. When the clamp was adjusted it was found that the muscle bundle of His alone was involved in the compression if the experiment was accurately performed. The amount of pressure could be regulated by means of a thumb-screw and complete or partial constriction could thus be established. The result of such compression was to produce first occasional failure of ventricular contraction; next, a ratio of the auricular to the ventricular beats of 2 to 1, 3 to 1, 4 to 1, and finally complete heart block in which the ventricles contracted independently of the auricular rhythm. Sometimes at the moment when complete block was established the ventricle ceased beating for periods varying from a few seconds up to fifty-five seconds. This phenomenon Erlanger explains as probably resulting from the fact that a certain time is necessary after complete severance of the connection between auricles and ventricles for the development in the ventricle of its inherent rhythmicity.

The experiments of Gaskell, His, Tigerstedt, Erlanger and others practically establish the fact that experimental heart block and the phenomena of Stokes-Adams' disease are identical. Up to the present communication, however, no case has been observed pathologically in which a lesion has been found involving the bundle of His. Dr. Erlanger tells me that when presenting his experimental work he suggested the possibility of involvement of His' bundle by a patch of sclerotic endocarditis beginning on the anterior mitral leaflet at its base and aortic border and extending to the interventricular septum where the bundle of His passes upward toward the auricle. The case about to be reported confirms the accuracy of his prediction.

J. B., aged fifty-seven years, a native of Germany, a wheelwright, was referred to the Out-patient Department of the University Hospital by Dr. B. F. Wentz, and to the medical ward by Dr. H. D. Jump. The patient was a married man, the father of five children, two of whom died in infancy: one of pneumonia, the other

of measles. He had been a healthy man, the only illnesses remembered by him being slight attacks of influenza, malaria, and lumbago. He denied venereal history, and there was no evidence of any such infection. He had never had any severe injury to the head or other parts of the body. He used tobacco in moderation, but had drunk beer to excess until two years ago, after which time he was very moderate in its use. The fatal illness began without warning while at his work about two and a half years ago. He suddenly fell backward and became unconscious for a few moments. There were no convulsive movements and he did not bite his tongue. The bladder was not emptied. He very speedily recovered from this attack and went back to work. Three months later he had another exactly similar attack and then the attacks came on in more rapid succession, about every two or three weeks. Except in the first attack he said he always fell forward. Two years ago he sometimes had spasmodic movements in the legs during the attacks, but the seizures were of varying severity, sometimes being extremely mild, when the patient did not fall nor lose consciousness but merely became slightly giddy for a few moments. In such mild attacks he was able by holding to some object to steady himself and prevent his falling. He found that stooping was more likely to provoke the attacks than any other movement or exercise. He has been troubled with flatulence after eating and has had obstinate constipation. Sometimes the attacks came on while eating, and occasionally belching would prevent their occurrence. At times there was pain in the head before the attack was fully developed and less severe headache was almost constantly present. He noticed that his face became cyanosed before the attacks and his friends observed that the cyanosis was intense during the attacks. After the seizure there was always violent palpitation.

The patient stopped working in May, 1905, and went to bed. During June there was a series of attacks, one following another every few minutes. Later they were less frequent, but he continued to suffer with the attacks until his admission to the hospital. He has lost sixty pounds in weight and has grown steadily weaker. He never had œdema nor dyspnœa.

Condition on Admission. The patient was admitted to the University Hospital on September 19, 1905, when the following notes were recorded:

The patient is a fairly well-nourished man; no cyanosis nor jaundice. There is marked pulsation in the epigastrium and in the veins of the neck. There is also visible pulsation of the brachial arteries. Radial pulses equal and synchronous; rate 36 and the character of the pulse "slow" (pulsus tardus). The radial and brachial arteries are somewhat hard. The pulse tension is moderately increased.

No enlargement of superficial lymphatic glands.

Eyes, nose, mouth, and ears normal; tongue slightly coated; teeth in fair condition.

Chest somewhat increased in the anteroposterior diameter; intercostal angle wide. Moderate depressions above and below the clavicles. Decided pulsation in the supraclavicular fossæ. Chest expansion fair and equal on both sides.

Apex beat visible in the fifth space just outside the midclavicular line. The impulse is moderately strong and there is a systolic thrill accompanying the beat.

Vocal fremitus is slightly diminished over the entire chest.

Percussion. The pulmonary note is everywhere slightly hyperresonant. Heart outlines: from the third space above to the sixth rib below and from the midsternum on the right to a point just outside the midclavicular line on the left.

Auscultation. The breath sounds are moderately weak, the expiratory sound slightly prolonged. There are no rales.

A somewhat harsh systolic murmur is heard at the apex of the heart and transmitted to the axilla. It is also heard toward the base of the heart. The second pulmonary sound is not accentuated, but is hollow and distant in character.

Abdomen. Epigastric pulsation is marked. Liver dulness begins at the sixth space and extends to the costal margin. Splenic dulness is normal in extent and the spleen cannot be felt.

The extremities are normal in appearance; reflexes normal. Station, gait, and muscular co-ordination normal.

Examination of the Urine. Specific gravity 1018, straw color, clear; reaction acid; no albumin; no sugar. Microscopically, a few hyaline and granular casts and scattered leukocytes were found.

September 22, 1905. The patient's condition has remained the same as on admission. There have not been any attacks and he feels quite comfortable.

Blood Examination. Red blood corpuscles, 5,260,000; white blood corpuscles, 14,000; hæmoglobin, 85 per cent.

23*d*. The patient began to have attacks this morning, a number occurring during the day. They have been mild in character, beginning with a momentary pallor, then a flushing or cyanosis and slight tremor of the arms. After the attack the face is pale and gradually the color returns. During several of the attacks it was noticed that the pulse stopped beating for a few seconds, and during one attack the interval was fifteen seconds. After the attack the pulse rate increased for a short time, then subsided to a steady rate of from 16 to 18 per minute. The pulsation of the veins in the neck continued during as well as between the attacks at from 80 to 100.

No change has occurred in the physical signs since the attacks have begun.

24th. The attacks are continuing at about one-half hour intervals. The pulse during the day has been from 18 to 26 and the volume has been good. The character of the attacks has not changed. During the seizures there seems to be only partial unconsciousness lasting a few seconds. The tremor attending the attacks is very slight. The eyes sometimes roll or move rapidly from side to side or upward in the earlier part of the attack and become fixed in a staring position when the attacks subside. The patient is quite weak and very nervous.

25th. The patient's strength has improved, as the attacks have been much less frequent.

26th. The paroxysms have grown more frequent and more marked. The development of the seizures to-day has been in the following order: The face first becomes yellow or pallid and the lips cyanosed; the eyes become fixed and staring, then move upward and to the left; the pupils change from a moderately contracted to a somewhat dilated condition, then the arms become a little rigid and tremble. The legs move very slightly in the same fashion. The pulsation at the wrist stops a few minutes before or during the attacks, and during one of the seizures the interval was thirty seconds. The venous pulsations in the neck continue through the attack. After the paroxysm the face becomes moderately flushed; the pulse begins to beat strongly, the respirations become deeper and more rapid than during the attacks. Unconsciousness is to-day somewhat more complete and of longer duration than previously.

During the afternoon the patient began to vomit, the first vomiting occurring just before an attack. Before and during the beginning of every seizure in the afternoon the pulse at the wrist stopped for from fifteen to twenty-five seconds.

27th. The patient is weaker and is becoming slightly drowsy. He slept poorly during the night and vomited repeatedly. After midnight the attacks followed each other in rapid succession. The seizures this morning are very severe. During one the pulse stopped for *two minutes and ten seconds.* The convulsive movements always follow the cessation of the radial pulse, and it is noticed that the face always becomes flushed before the patient recovers consciousness. The following table shows the duration of the cessation of the radial pulse, the number of venous pulsations in the neck per minute, and the rate of the radial pulse immediately after the attacks and some time later:

10 22 A. M. Ventricular beat for five minutes between attacks 147 per minute.

	Cessation of pulse.	*Venous pulse in neck.*	*Radial pulse.*
10.27	45 sec.	144	122
			1 min. later 24
11.20	80 sec.	140	132
			30 sec. later 84
			1 min. later 50
			2 " " 22
12.03	50 sec.	118	100
			1 min. later 80
			1½ " " 24
12.10	2 min. 10 sec.	120	90
			1 min. later 40
			1½ " " 38
12.20	Several attacks in rapid succession.		
1.00 P. M.	50 sec.		96
			1½ min. later 20
1.30	1 min. 20 sec.		100
			1 min. later 40

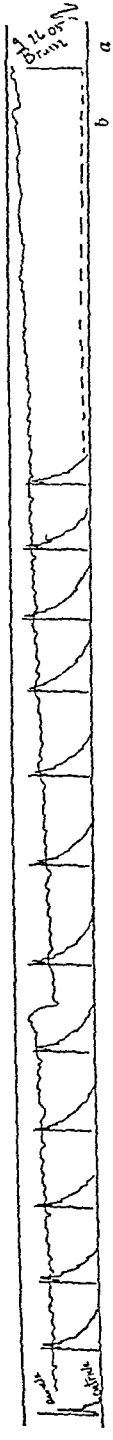
27*th*. The patient continued having repeated attacks of the same general character at short intervals. In the afternoon Cheyne-Stokes respiration began and shortly afterward the attacks ceased. Three hours later the patient died very quietly. The ventricular pulse remained low (18 to 26) to the last and the venous pulse in the neck continued rapid.

During the continuance of the severe paroxysms repeated examinations were made and confirmed the previous physical examinations. The systolic murmur heard at the apex was constantly present and the heart sounds corresponded accurately with the pulse felt at the wrist. Occasionally feeble heart sounds were heard and sometimes these coincided with slight evidence in the pulse tracing of weak systolic efforts. No suggestion of sounds corresponding to the venous pulsation could be heard over any part of the heart.

The accompanying pulse tracings were made with a tambour applied to the pulsating vein and a Jaquet's sphygmochronograph applied to the radial pulse (Fig. 1). In order to have the record of venous pulsation and arterial pulse on the same paper, the lever recording venous pulsations was of necessity a little behind that marking the arterial beats. The two records do not, therefore, present synchronous pulsations at any given point.

During the last few days of life a condition of complete heart block was seemingly present. The auricular (venous) and ventricular pulsations were entirely independent. Between the paroxysms the pulse rate was from 18 to 26 and the venous rate from 80 to 140. The latter rate was sustained during and between the seizures, while the pulse beats ceased for periods varying from twenty seconds to two minutes and ten seconds just before attacks and rose to from 90 to 140 immediately after the seizure. In the intervals the radial pulse was regular and fairly strong, the rate being from 18 to 20 or 30 (Fig. 2). The blood pressure was estimated on one or two occasions between attacks and was found

FIG. 1



Tracings of venous (jugular) and arterial (radial) pulses taken simultaneously. In the large tracing is shown a long pause, during which the ventricle stopped beating, the auricular (venous) pulse, however, continuing though the waves became less and less up to the moment of the first succeeding ventricular beat.

a. Down stroke of this wave is omitted.

b. A small contraction wave at this point is omitted.

135 mm. systolic and 65 diastolic. (The figures are probably inaccurate, as the observation was made with some difficulty.)

Various diffusible and other cardiac stimulants were administered without effect. Ammonia, atropine, strophanthus, and nitroglycerin were used, but none had the slightest effect on the pulse rate. The venous rate increased, but whether as a result of the medication or of increasing weakness could not be determined. During the earlier days of his residence in the hospital it was observed that ordinary exercise had no effect on the pulse rate.

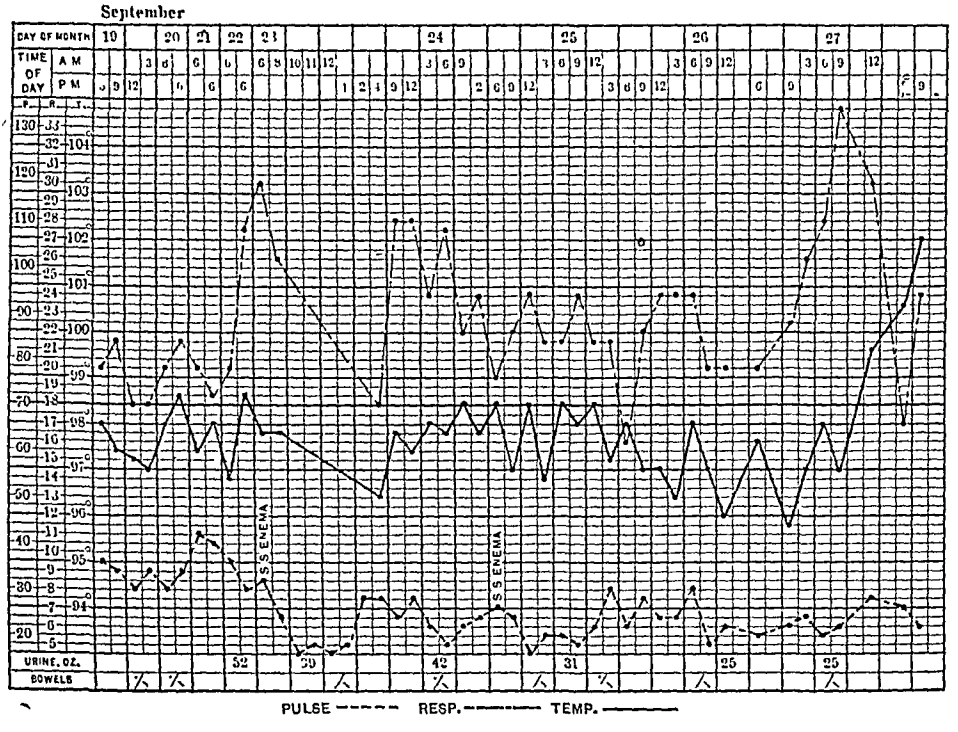
Some authors have insisted that the venous pulsations are positive pulsations transmitted from the right ventricle through an incompetent tricuspid valve, the ventricular beat being so feeble that the aortic valve remains closed or the pulse at the wrist is so weak that it fails to become palpable. The tracings taken in my case seem to disprove this view. The venous pulsations continued without notable change and without any relation to the regularly recurring and forcible ventricular pulses during the intervals between attacks, while in the long pauses between radial pulses the auricular waves grew somewhat less forcible toward the end of the pauses, as might be expected when the ventricle was at rest.

Autopsy. The conditions found at autopsy will be only briefly described. There was a moderate grade of general arteriosclerosis. The heart was hypertrophied, the walls of the left ventricle being especially involved. The heart muscle was entirely normal in appearance in all parts except where an endocardial lesion affected a small area at the upper part of the interventricular septum. The root of the aorta was slightly atheromatous, and the openings of both coronary arteries were involved. The coronary arteries themselves were distinctly thickened, but in no place calcareous or stenotic.

Their lumen was enlarged if at all altered. The aortic leaflets were slightly diseased but not calcareous. The valve seemed to be competent.

The lesion of importance was one on the anterior mitral leaflet toward its base and aortic edge. This was a patch of atheromatous character, sclerotic and white, and it extended to the endocardium exactly over the bundle of His where this band passes from the ventricle to the auricle. The relations were determined by removal

FIG. 2



Record of pulse, respirations, and temperature.

of the endocardium at the upper part of the interventricular septum. Viewed from the side of the right ventricle the specimen does not show any evidence of penetration of the sclerotic process, but on the left ventricular side it is evident that the bundle of His was involved in the area of disease.

The exact extent and relations of the pathological process cannot be stated until the histological studies now in course of completion are finished. These will be published in a subsequent paper.

The important fact in connection with the case is that the heart muscle was unaffected by any degeneration or sclerotic change excepting in the very limited area of the middle portion of the auriculoventricular bundle.

REVIEWS.

MANUAL OF ANTENATAL PATHOLOGY AND HYGIENE. THE FETUS. By J. W. BALLANTYNE, M.D., F.R.C.P.E., F.R.S.Edin., Lecturer on Midwifery and Gynecology, Medical College for Women, Edinburgh; Lecturer on Antenatal Pathology and Teratology in the University of Edinburgh (1900); Examiner in Midwifery in the University of Edinburgh; Assistant Physician, Royal Maternity Hospital, Edinburgh; Honorary Fellow of the Glasgow Obstetrical and Gynecological Society, and of the American Association of Obstetricians and Gynecologists. Edinburgh: William Green & Sons, 1902.

THE name of the author of this valuable book has become familiar through his studies in the difficult field of embryonal and fetal pathology. For this he has had especial advantages in being able to examine large numbers of specimens, while his consecutive study of the subject has added constantly to the interest and accuracy of his observations.

The work under discussion begins with a definition of the relation existing between antenatal and postnatal pathology. The author considers the antenatal factor in gynecological pathology and finds in this a reason, if possible, of such disorders. He draws attention to the effect of traumatism in birth and describes fully what he terms the physiological readjustment which takes place after birth.

Under the head of intranatal traumatism the author describes cephalhæmatoma, facial paralysis, fractures, and dislocations. In treating intranatal infections he believes that gonorrhœal ophthalmia should be anticipated and prevented by treatment during pregnancy. In our observation such treatment can be completely successful, although the mother's gonorrhœa may be of the most pronounced and virulent type. In the author's observation the absence of vernix caseosa has seemed to expose the infant to increased danger of mastitis.

Under the title "Scourge of St. Kilda" an interesting description is given of tetanus of the newborn in this island, and the tracing of its origin to infection of the umbilical cord; when antiseptic measures were employed in treating the umbilicus the scourge ceased. The author calls attention to the fact that the germs which produce septic infection in the newborn are everywhere present, and that

it is not remarkable that such infection arises, but rather strange that it does not more often happen. He urges thorough general precautions in the care of all infants. The etiology of hæmoglobinuria remains to the author in doubt, although the evidence of infection in these cases predominates. Under the head of Omphalorrhagia the author describes what seems to us septic infection of the umbilicus. This might have been to greater advantage described in connection with other septic processes.

Under the title of Neonatal Readjustments the author describes icterus, melæna, keratolysis, pemphigus, and sclerema.

In considering the pathology and hygiene of the fetus the author calls attention to the neofetal period during which the placenta is being established. He describes the growth of the fetus as most active if weighed between the fourth and fifth months, when the placenta is being fully established; placental weight corresponds to fetal weight. The author adds a scheme and diagram illustrating the development of the various organs.

In considering the anatomy of the fetus illustrations are given of sections through the cranium, thorax, abdomen, and pelvis; many of these are in colors; while they are not remarkable for excellence as illustrations, they aid considerably in elucidating the text.

The author describes the syncytium as a layer of nucleated protoplasm covering the villi of the full term placenta.

The fetal circulation receives full discussion, and the blood of the child is described at considerable length. The excess of leukocytes in the blood coming from the mother indicates a migration of white corpuscles from the maternal to the fetal blood, which may be a phase of fetal nutrition. In describing fœtal respiration, respiratory movements are recognized as occurring in utero.

The chemistry of the placenta and of the fetal excretions receives adequate treatment, while the functions of the ductless glands of the fetus are described.

In fetal pathology the author makes a minute classification, commencing with those disorders which are transmitted from the mother. He rightly emphasizes the importance of the placenta and membranes in studying the fetal body. A given specimen is certainly incomplete without fetus and membranes, and no autopsy upon the fetus alone without examination of these important structures can be conclusive. The infections transmitted from mother to child are fully described and every possibility considered is fully demonstrated. While fetal tuberculosis is rare, the author believes that about twelve instances of tuberculosis without lesion are recorded. The absence of tuberculous infection arises from the successful barriers to infection afforded by the placenta and the fetal liver. The author believes that tuberculous parents frequently have children who are malformed and cites the instance of a child born of a tuberculous mother who had web toes. Rheu-

matic fever is considered as one of the fetal diseases arising from transmission from the mother.

The author discusses fetal syphilis very fully and considers Colles' law by no means absolute.

The toxicology of fetal disease is fully considered, and among other causes of fetal mortality working in tobacco is recognized.

The author believes it is possible for the mother to be seriously ill with diabetes, cancer, leukæmia, or other diseases, and even eclampsia, and for the fetus to be born living and well. He considers that it is exceptional to find the morbid maternal state reproduced in the fetus; here we cannot agree with him, as we have seen fetal deaths from toxæmia follow two weeks after the birth of a vigorous child from an eclamptic mother. The cystic diseases of the fetus and elephantiasis are grouped together as illustrating diseases of the connective tissue. The diseases of the fetal skin are described at length with illustrations.

The writer discusses diseases of the fetal skeleton, discussing some of the rarer forms and omitting some conditions which are not infrequent, such, for example, as rachitis in the fetus. Fetal ascites is fully described and illustrated and attention drawn to the condition as a symptom of important pathological states in the viscera of digestion. In discussing the heart and its disorders, atheroma and endocarditis are referred to transmitted infections.

In considering disorders of the pelvic viscera, the writer believes that there is a downward displacement of the uterus which frequently occurs soon after or at birth and which is distinct from elongation of the cervix.

In concluding the volume the author describes briefly the accidents and injuries sometimes seen at birth and gives ample space to the discussion of hydramnios. Fetal death is fully described and the possibility of fetal rigor mortis is admitted. A colored illustration shows some of the changes which the fetus undergoes during maceration. As regards treatment of fetal death, if the membranes are unruptured, the physician is advised to wait, allowing changes in the placenta, membranes, and uterine wall to take place, which will diminish the chance of sepsis and hemorrhage. If the membranes are ruptured, the indications for interference are more clear, but even then it is wiser, keeping the patient under observation, to wait until the beginning of uterine contractions.

While the use of the antepartum douche may be permitted, it must be employed with great caution.

The consideration of the symptoms and diagnosis of fetal conditions and a consideration of those measures best adapted to promoting a sound and healthy development of the fetus conclude the volume.

This excellent work gives all the light available upon one of the darkest portions of pathology. Its practical value is great, for it

explains many phenomena not uncommonly observed in obstetric practice, and gives a rational basis for some important decisions regarding the treatment of the mother in the interest of the child. No one can rightly decide upon the various obstetric operations whose purpose is to save the life of the child without understanding sufficient of the conditions which make this life possible to choose wisely. While obstetric surgery is rapidly advancing and obstetric operations are more and more successful, they will frequently fail unless performed in cases where the child is in good condition. To recognize the fetal condition a knowledge of its physiology and pathology is of the greatest value.

It was our purpose to review with this the author's more recent volume on the pathology and hygiene of the embryo, which completes the entire subject, but this journal has not received this volume for review. E. P. D.

L'OEUF HUMAIN ET LES PREMIERS STADES DE SON DEVELOPPEMENT. Element d'embryogene par T. POTOCKI et A. BRANCA. Avec 100 figures et 7 planches. Paris: G. Steinheil, editeur, 1905.

THIS book of 196 pages has been written to serve as an introduction to the study of obstetrics, and does not pretend to be a treatise on embryology. The authors start with the intention of following the development of the ovum and of the fetal membranes in man alone. The great difficulty in such a study of making certain points clear without reference to comparative embryology is immediately apparent; so that even with their fixed purpose in view the authors continually revert to the development of the lower animals in describing the changes in growth of the human embryo. But since these references to comparative embryology are curt and introduced rather apologetically one feels only half enlightened upon certain parts of the subject. In their treatment the authors consider in turn "the sexual products," their origin and their morphological value; "the genital apparatus and the sexual life," ovulation and the corpus luteum, menstruation, the genital organs during pregnancy, the genital apparatus, and the postpartum; "the first stages of development of the embryo," fecundation, the segmentation of the egg, the formation of the embryonic membranes; "the embryonic membranes," the umbilical vesicle, the amnion, the allantois, the chorion, the fixation of the ovum and placentation, the umbilical cord. The book is well illustrated with figures, and contains seven rather familiar but well-executed plates representing in a schematic form the development of the fetal membranes. By reason of the large print and short paragraphing, so typical of the French, the book makes easy reading, and is on the whole interesting as an introduction to obstetrics. W. T. L.

STUDIES IN GENERAL PHYSIOLOGY. By JACQUES LOEB, formerly of the Department of Physiology, now Professor of Physiology in the University of California. The Decennial Publications, second series, Vol. XV. Chicago: The University of Chicago Press, 1905.

THE decennial publications from the University of Chicago are now well known to the general medical public. The present volume comprises a notable series of papers. Professor Loeb's unusual work in physiology has had wide recognition, and most of the articles which have been gathered together to form these volumes are already familiar to many whose immediate line of investigation lies outside of physiology. Practically all of Professor Loeb's original contributions have been collected and bound together to form the two volumes. There are thirty-eight papers, a large number of which appeared originally in German, and have been translated for this publication. In the first volume are the earlier articles dealing with the subject of heleotropism, while in the second volume one finds the well-known experiments on artificial pathogenesis. It is not, however, necessary to discuss the various subjects in detail. It is highly satisfactory to have these papers put into an accessible form, and for this one is indebted to the decennial publications. W. T. L.

ANATOMIE DES MEMBRES. DISSECTION. ANATOMIE TOPOGRAPHIQUE Par le DR. CH. DUJARIER, ancien prosecteur à l'Amphithèatre de Clamart, chef de clinique chirurgicale à la faculté. Avec 58, planches originales dessinées par Reignier. G. Steinheil, editeur, Paris, 1904.

UNDER this title M. Dujarier publishes an atlas which is "the fruit of his years as prosector in the amphitheatre of anatomy of Clamart." The book is intended for guidance of students in the dissecting room, placing beneath their eyes types of anatomical preparations which have been drawn by Reignier from specimens dissected by the author. There are 58 original plates, mostly in colors, illustrating the different steps in the dissection of the various parts of the limbs. Anomalies which are frequently encountered have been inserted. In addition to these plates there are 19 taken from Farabeuf. In the text each region is described with a little more detail than is usual in an atlas in order that the student will not be obliged to have recourse to a descriptive anatomy for a complete account of the structures. The author has particularly developed the chapters on the axilla, fold of the elbow, palm of the hand, region of the buttock, Scarpa's triangle, knee-joint, and sole of the foot. The book is clear and accurate and is recommended not only to students, but also to physicians who may at a glance desire to refresh their memory of the anatomy of the extremities. F. T. S.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Meningismus Typhosus and Meningotyphus.—STAUBLI (*Deutsch. Arch. f. klin. Med.*, 1904, Band lxxxii. p. 90). Curschmann, in 1886, published 5 cases of genuine purulent meningitis complicating typhoid fever, and in his article draws attention especially to cases of typhoid setting in with signs of meningeal irritation, which, however, at the end of a week or two recede to give place to the usual symptoms of the disease. Fritz had previously noted similar cases and had pointed out that occasionally the meningeal symptoms persisted until late in the disease. He also emphasized the fact that such cases dying and coming to autopsy showed no lesion of the cord or brain. Since then many authors have commented on the condition, and in 1888 Wolf published 12 cases from Curschmann's clinic, 3 of which died, yet at autopsy the brain and cord were found to be normal, while cultures from the central nervous system were negative. Such instances have been explained by the presence of a soluble toxin with special affinity for the nerve cells. Stäubli cites 2 cases from Müller's clinic. In the first, with marked meningeal symptoms, no organisms were obtained by cultures from the spinal fluid, and after death the central nervous system showed no lesions. In the second there were, besides meningeal symptoms, motor cortical aphasia and ataxia of the legs and arms. This patient made a complete recovery. For such cases Bouchut has suggested the name "meningismus typhosus."

There are, however, cases in which at autopsy a purulent meningitis is found to explain the symptoms. Birch-Hirschfeld points out that brains showing apparently only a congestive hyperæmia may, on microscopic examination, display the early changes of inflammation. It was

for a long time contended that the typhoid bacillus could not produce a purulent inflammation and that the meningitis when present was caused by a secondary infection. There are now, however, numerous instances where the typhoid bacillus alone has been cultivated from the spinal fluid obtained during life and from the exudate after death. It would not be surprising if one could occasionally obtain typhoid bacilli from the brain, considering how widely distributed they are in the body, and to render the evidence conclusive they should be demonstrated in the exudate in sections. Stäubli offers such convincing evidence in a case he reports.

Enterogenous Cyanosis.—HIJMANS V. D. BERGH (*Deutsch. Arch. f. klin. Med.*, 1905, Band lxxxiii. p. 86). In 1902 Stokvis described a new clinical picture under the name "Autotoxische Enterogene Cyanose." His patient, a man, aged thirty-eight years, had severe enteritis, clubbed fingers, and a very remarkable degree of cyanosis of the skin and mucous membranes, spectroscopic examination of the mucous membranes showing definite methæmoglobin bands. The similarity of the condition to poisoning with certain drugs, as with potassium chlorate and the aniline derivatives, led Stokvis to assume the development in the diseased bowel of some poisonous substance, which, when absorbed, converted part of the hæmoglobin into methæmoglobin. In the same year Talma reported 3 more cases, all with diarrhœa, clubbed fingers, intense cyanosis of the skin, and mucous membranes and methæmoglobinæmia. He demonstrated the characteristic spectral bands in the blood taken from the finger, as well as in the skin and mucous membranes, and showed further that the methæmoglobin was not free in the plasma, but remained attached to the corpuscles. To these observations v. d. Bergh adds two of his own.

His first case is that of a boy, aged nine years, who was born with atresia ani, which was operated upon. At the end of several months a rectourethral fistula formed, and since then has remained patent, so that feces and urine are both emptied through the bowel. For four or five years the mother had noticed that on exertion the child became cyanosed, and for a year previous to admission he had been blue, even while at rest. The poorly developed, emaciated child showed an intense cyanosis, which varied from day to day, but was always deeper after exertion. Heart and lungs showed no evidence of disease. In the rectum was a fibrous stricture, and above this a rectourethral fistula. At times fecal material could be pressed from the penis, but by catheterization urine was obtained, which was pale, cloudy, free from albumin and sugar, but contained much indol. Stools were frequent, thin, alkaline, with strong ammoniacal odor, and turned black on exposure to air. Spectroscopic examination showed the presence of a substance giving a band in the red, which was at first assumed to be methæmoglobin.

Considering such cases as evidently of toxic origin, attempts have been made to isolate the poison in the urine. Stokvis found that the urine after undergoing alkaline decomposition turned a deep red color on the addition of mineral acid, and rapidly converted oxyhæmoglobin into methæmoglobin, properties which the voided acid urine did not

have. Steensma has since shown that all urines which have stood any length of time give the color reaction above noted and convert hæmoglobin into methæmoglobin through the formation of nitrites, while the addition of a small amount of nitrite to fresh urine gives it these properties at once.

Oxyhæmoglobin converted by the urine of v. d. Bergh's case gave also a typical methæmoglobin band, but, to his surprise, on careful comparison, the band from the blood was placed a little to one side of this. Portions of the patient's stool mixed with normal blood gave this band, and v. d. Bergh concluded that the substance could not be true methæmoglobin and that the poison, whatever it may be, is not eliminated in the urine. After trying the action of numerous chemical agents on normal oxyhæmoglobin, he found that only hydrogen sulphide gas would produce a substance with an identical spectroscopic picture. Cultures from the stools showed a hydrogen sulphide producing bacterium, and this planted in a normal oxyhæmoglobin solution caused the same change. The effects of hydrogen sulphide gas on oxyhæmoglobin had previously been carefully studied by Hoppe-Seyler and Harnack, and the substance formed was called sulphohæmoglobin. This gives a distinctive spectroscopic picture which no other hæmoglobin derivative simulates, and corresponds exactly with that produced by the blood of the patient.

There is nothing in the literature concerning autointoxication from the gradual absorption of hydrogen sulphide from the intestines. While Bergeon's method of inflating the rectum of tuberculous patients with hydrogen sulphide gas was in vogue no spectroscopic examinations of blood were made. In animals the administration of Schlipp's salts causes sulphohæmoglobinæmia, while inhalation of the gas does not. Meyer has poisoned rabbits by directing a flow of H_2S into the intestines. It is difficult to see why in this special case there should have been any absorption of hydrogen sulphide with poisoning, and why in so many others where the gas is formed it should lead to no toxic symptoms. v. d. Bergh points out that the two most important factors in abnormal absorption were present, stagnation of intestinal contents and dilatation of the intestinal walls.

The second case reported is one of true methæmoglobinæmia. The patient, aged twenty-five years, was admitted for acute nephritis. He presented an intense grade of cyanosis, which had come on suddenly seven years before. His blood gave the typical reaction for methæmoglobinæmia. Intestinal symptoms were much milder than in previously reported cases. The cyanosis, too, was very variable, always deeper in the evening, irrespective of rest on exertion, and showing a remarkable relation to diet. An absolute milk diet for two days caused the cyanosis to disappear entirely, only to return with its former intensity after the first mixed meal. Meat seemed to exercise the strongest influence upon it.

SURGERY.

UNDER THE CHARGE OF

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Some Surgical Notes on Tuberculosis of the Kidney.—KELLY (*British Medical Journal*, June 17, 1905, p. 1319) has never seen a case of primary ascending tuberculosis in upward of 50 surgical cases, for the most part in women; although he has seen several cases of vesical tuberculosis, primary or associated with a genital tuberculosis, but the kidneys were not involved. He has also seen coincident renal and genital tuberculosis, but without other manifest connection than the vascular system or some remote depot of infection. Without any doubt the infection of the kidney is almost always hæmatogenous, the primary focus being often in the thorax, sometimes in a cheesy bronchial gland apparently dormant. Therefore, in searching for evidence of latent disease, the lungs should always be examined carefully, and the region of the lymphatic glands of the neck should be searched for swelling and scars, as well as joints for old troubles. Tuberculosis once started in the kidney is almost always progressive, a spontaneous cure being exceedingly rare.

The real crux of the situation is the diagnosis. It is satisfactorily made when tubercle bacilli are found in the urine at more than one examination, and preferably by more than one observer, and when these are traced upward to the kidney and positively excluded from the other. In all obscure cases two guinea-pigs should be injected, one into the peritoneum, the other into the axilla. In three weeks the disease will have developed if present. The injection of tuberculin is valuable if, in addition to the general reaction, there is a marked local reaction in the form of pain in the kidney. A practised examiner will, in these early cases, find a slight thickening or hardening of the ureter on one side. An inspection of the ureteral orifices will often disclose a tell-tale blush or puffiness or granular condition. Any slight persistent cystitis rebellious to treatment should always be suspected as tuberculosis, and in such a case the diagnosis must be looked upon as incomplete until the condition of the kidney has been investigated. Cystoscopic examination is very valuable, often showing an ulcerated, suppurating, bleeding bladder. Involvement of only one ureteral orifice promises well, and renders useless the catheterization of the sound ureter, although Kelly has never seen any harm come from it.

Radical treatment, the extirpation of the disease in every case that will permit it, is the rule; but the other kidney must have been proven capable of sustaining life by a thorough analysis of the separated

urines. The diseased area should be excised only in rare cases in which it was evidently limited to one pole, as demonstrated by splitting the kidney from pole to pole and down to the pelvis, as it lay outside the abdomen before ligation and detachment. Nephrectomy, except as a preliminary to a radical operation, has proven to be an operation of necessity, not of election. When it is performed, all abscesses in both poles of the kidney should be opened.

Caution is advised before adding the ureteral extirpation to that of the kidney. The cases best adapted to the radical operation are those with a greatly enlarged, often strictured ureter. Involvement of the vesical walls has always been deemed one of the most serious local extensions of the disease, and by a number of surgeons is considered beyond the pale of surgical relief. The steps to be followed in these bad cases are: 1. Drain a bladder thoroughly. 2. Then remove kidney and ureter. 3. Use the prolonged bath at even temperature of 100° for hours or all day. 4. Close the fistula. 5. Treat any remaining small patches of disease with nitrate of silver, applied through the open-air speculum. 6. Use distention and irrigation treatments. 7. Excise any rebellious areas of the disease.

The small patches of disease around the ureteral orifice are rarely tuberculous, and this is the reason why they clear up readily without further treatment after nephrectomy.

Concerning the Subcutaneous Transference of the Omentum.—NARATH (*Zentralblatt f. Chirurgie*, August 12, 1905) says that the last word has not been said of the Talma operation. While it is doubtful whether the operation has influenced favorably the diseased process in the liver itself, it is certain that it is followed sooner or later by a disappearance of the ascites and some of the disturbances of which the patients complain, and that the patients feel well again and are able to work. Of the abdominal organs which can be fixed to the abdominal wall, the omentum and spleen deserve special mention. The cases in which the spleen can be sutured to the abdominal wall will not be particularly numerous, since we do not care to burden with further disturbances patients who are already very miserable.

The fixation of the omentum has been attempted by very different methods. One should give that method the preference which can be carried out most simply and without narcosis. Narath has concluded after repeated trials that it is best to suture the omentum in a subcutaneous pouch in the anterior abdominal wall. He says that this method produces an abundant anastomosis.

The technique is as follows: 1. Under Schleich's method of local anæsthesia the abdomen is opened in the median line, just above the umbilicus, to the left of the round ligament, thus sparing the paraumbilical veins as much as possible. 2. The abdominal incision should not be unnecessarily large, lest the intestine escape through the wound alongside the omentum. The ascitic fluid should be thoroughly evacuated from all the dependent parts, siphoning it out by tubing if necessary. 3. A thick end of the great omentum is then seized and drawn out of the wound without disturbing the transverse colon from its natural position. This part of the omentum should contain as many large bloodvessels as possible, and at the level of the umbilicus should be of one to two fingers' thickness. 4. The abdominal wall should now

be closed about the omental stump without compressing the omentum, which should come through in a direct line. 5. The omentum is then fixed to the parietal peritoneum and linea alba by a few fine sutures, avoiding the veins. 6. The omentum outside the abdomen is then lodged in an oblique pouch in the subcutaneous tissues, about 15 cm. long, and directed to the left of the median line. 7. A water-tight closure of the skin wound should be made and a bandage applied that will not compress the subcutaneously situated omentum.

In the next few days there is observed an œdema of the abdominal skin, probably due to an escape of ascitic fluid. Later this disappears, and in about eight days a radiating group of small veins are seen, while the larger veins increase in size. The displaced omentum becomes distinctly swollen and the veins filled to their utmost. The degree of venous dilatation differs individually very much. Narath has seen some veins in the abdominal wall swollen to the size of the thumb.

An occasional danger comes from the slipping alongside the omentum through the abdominal incision of a portion of the transverse colon or small intestine to a subcutaneous position. This is due to faulty technique in the closing of the wound about the stalk of the omentum.

THERAPEUTICS.

UNDER THE CHARGE OF

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Absorption, Elimination, and Mode of Action of the Quinine Salts.—DR. ARMAUD has concluded from observations of the physiological action and elimination through the urinary tract that, in individuals without febrile movement, quinine is present in the circulation in from six to nine hours after administration; in febrile conditions it is absorbed after from nine to twelve hours. The dosage being equal, elimination takes place more rapidly in subjects who have taken the drug for some time, since the organism, already saturated, throws off the greater part of the quinine ingested; consequently the greater the dosage the more of the drug will be eliminated. Reduction of the dose renders assimilation more free, retards its elimination, and reduces the quantity of quinine in the free circulation. The therapeutic action depends rather upon the amount of the drug in the circulation than upon the quantity assimilated by the tissues. The action of quinine, then, should depend upon the amount taken and its maximum effect coincide with the presence of the maximum amount free in the blood current; that is,

about six to nine hours after ingestion in the afebrile individual and from nine to twelve hours in fever patients.—*Marseille médicale*, 1905, No. 11, p. 356.

Thiosinamine Injection in Cicatricial Œsophageal Stricture.—Dr. Karl Schneider reports a case of cicatricial stricture of the œsophagus following the ingestion of caustic soda in a child of five years; the œsophagus of the patient after the initial inflammation was healed would allow the passage of a No. 24 (F.) sound. The child was not seen for five months, and at the next observation the obstruction prevented the passage of the finest bougie. Injections of thiosinamine, 1 part; glycerin, 4 parts; water, 16 parts; 10 minims were prescribed. After two injections three days apart a No. 9 (F.) sound could easily be passed. The quantity of the injection was raised to 15 minims, the treatment was continued, and in about two weeks the stricture would admit a No. 28 (F.) sound.—*Correspondenz-Blatt f. Schweizer Aerzte*, 1905, No. 11, p. 356.

Aristochine in Whooping-cough.—Dr. H. Koppe advocates the employment of this drug in pertussis. At first it is administered only once or twice daily, but after a few days the patient should receive from three to five doses during the twenty-four hours. The dosage for children under a year old is from 3 to 4 grains; for those beyond this age 7½ grains at a time, given in sugar or with the pulp of fruit; a short time afterward the patient should receive a few drops of diluted hydrochloric acid well diluted with water (this should be taken through a tube to save the teeth); the author has never known the drug to cause unpleasant sequelæ.—*Deutsche Aerzte-Zeitung*, 1905, No. 9, p. 201.

The Treatment of Capillary Bronchitis in Children.—Dr. Schopohl advocates the employment of baths at 96.8° to 100.4°, increased gradually to 104° to 113°, and lasting ten to twenty minutes. To maintain the resulting cutaneous congestion the patient is wrapped, without being dried, in a sheet; outside this a covering of linen fabric is applied and the child allowed to rest for an hour or two; he is then unwrapped and the chest enveloped in oil silk, the dressed side of which has been smeared with warm oil. One such procedure usually suffices to greatly improve the patient's condition, but in severe cases it may be necessary to employ it twice during the same day. As an adjunct to this treatment the continuous inhalation of steam is recommended. The author considers that his method lessens the intrapulmonary congestion, increases the excretion of urine, relieves the overacting heart, and brings about a general improvement.—*Blätter f. klin. Hydrotherapie*, 1905, No. 4, p. 80.

Hereditary Syphilis in Infants.—Dr. B. Salge considers the prognosis favorable in those cases in which symptoms, coryza, etc., are manifested six weeks or so after birth. He disapproves of the treatment by baths in bichloride solution on the ground that there is no means of ascertaining how much of the drug is absorbed and because their action is not rapid unless the skin is scarified. He considers inunctions and the internal administration of mercury, in the form of calomel or the yellow iodide, preferable. The most efficacious method, however, is, in his

opinion, that by hypodermic injection. He uses the following formula: Mercury bichloride and sodium chloride, of each 0.2 part; water, 10 parts. Of this solution one-tenth of the content of a Pravaz syringe is injected into the substance of the muscles once a week. The treatment is but slightly painful and is very efficacious. For the syphilitic coryza the author employs insufflations of 1 part of calomel to 20 parts of sugar, applications of red mercuric oxide upon bits of cotton, and a spray of adrenalin chloride. If bloody stools with mucus occur the treatment is stopped. The fact that specific disease interferes to a marked degree with the nutrition and development of the child makes energetic treatment necessary. The preferable food for these children is, of course, breast milk.—*Berliner klin. Wochenschrift*, 1905, No. 22, p. 690.

Marmorek's Serum in Tuberculosis.—DR. T. STEPHANIE has used this substance in the treatment of 7 cases, in 2 of which he achieved rather remarkable results. He concludes that in this remedy we have a powerful agent in the treatment of tuberculous conditions, both intrapulmonary and extrapulmonary, and one which is, in general, harmless. Not only is it potent in recent cases, but in those of long standing as well, in which other methods of treatment have proved of little avail. He expects to continue its use in his practice and believes those in such diseases as that due to the bacillus of Koch, while our resources in the combat against them are all too few, we should omit no form of treatment which may possibly result in benefit to the patient.—*Le Progrès médicale*, 1905, No. 25, p. 393.

The Treatment of Jaundice.—DRS. H. HUCHARD and C. FIESSINGER vary the treatment of this condition in accordance with its causation; in all cases, however, a milk diet is necessary—a tumblerful every four hours, with the addition of a little alkaline water, lime-water or Vichy. The alkaline mineral waters and infusions, such as aniseed tea, may be allowed. The bowels should be opened by sodium sulphate or other mild alkaline; calomel is neither effectual nor well borne; intestinal antiseptics, salol, naphthol, etc., should not be given. Intestinal irrigations, when there is intestinal colic, are indicated; a quart of hot or cold water may be given in this manner morning and evening. The saline laxative should be continued for a week, then omitted for a week, during which 15 grains of sodium benzoate or salicylate are administered at noon and evening. The morning laxative may be replaced by a pill of euonymin, gr. $\frac{3}{4}$; podophyllin and belladonna extract, each gr. $\frac{1}{6}$. The milk diet should be continued, and to it may be added a little macaroni or vegetable purée soup. In tertiary syphilitic jaundice specific treatment is indicated. Pruritus may be relieved by alkaline baths, warm douches, a warm lotion of coal tar, $\frac{1}{2}$ ounce to the quart of water, weak solutions of carbolic acid, mercury bichloride, 1 : 2000, ichthyol, mentholated talcum powder, etc. If it is obstinate the skin may be dressed with ichthyol, 10 parts; alcohol and ether, each 50 parts, followed by a soothing powder, or by glycerin, 20 parts, to chloroform, 60 parts. In icterus due to gallstones, a course of spa treatment may prove effectual, or this failing, surgical intervention may be considered. If symptoms of cholæmia appear the milk diet is necessary and an enema should be given each morning, to be followed after an hour by an irrigation of 8

ounces of saline solution; about 3 ounces of macerated pork liver should be given daily mixed with a little water. The nervous symptoms may be relieved by packing in a sheet wrung out in water at 100.5° twice a day or by two or three baths daily at from 82° to 86° and lasting ten or fifteen minutes.—*Journal des praticiens*, 1905, No. 23, p. 361.

OBSTETRICS.

UNDER THE CHARGE OF

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Puerperal Endocarditis.—In the *Monatsschrift für Geburtshülfe*, 1905, Band xxi., Heft 5, WINKLER reports the case of a patient who was delivered of a living child in spontaneous labor; the afterbirth was also expelled spontaneously. Fourteen days after labor the patient had fever, with swelling of the joints of the right hand and then of the right foot. An eruption appeared on the face, which spread over the entire head. The patient was admitted to the hospital and was treated by inunction and developed the characteristic temperature of pyæmia. The pelvic organs were normal, but endocarditis developed. The patient developed cyanosis, with high fever, chills, and died in collapse.

Upon autopsy the pelvic organs were normal, but endocarditis was fully developed. In the joints were found foci of infection. The exciting cause of the infection and the nature of the infective germ were not discovered.

Ileus in the Puerperal Period Successfully Treated by Eserin.—KREBS (*Monatsschrift für Geburtshülfe und Gynäkologie*, 1905, Band xxi., Heft 5) reports the case of a multipara, who, three and a half days after a normal labor, developed abdominal distention and vomiting of bile, and normal pulse and temperature. There was no hernia, and the uterus was not retroverted, nor could a tumor be found. During the last months of her pregnancy the patient had vomited frequently.

An affort was made to move the bowels by injection, but without success; 1 mg. of eserin salicylate was given hypodermically. Half an hour later peristalsis began, gas escaped from the bowel, and three-quarters of an hour later the patient's bowels moved several times. She made a good recovery.

The Nature of Hæmatoma of the Muscles of the Neck in the Newborn.—In the *Zentralblatt für Gynäkologie*, 1905, No. 20, PINCUS reports the case of a child born in spontaneous labor who died fourteen days after birth. There was no history of syphilis in the parents. Two days after delivery the child developed on the left side of the neck a hæmatoma

of the sternocleidomastoid muscle. On microscopic examination of this tumor, a fibrous myositis was present. This evidently was of a progressive nature, and would have involved a considerable portion of the muscle. It was interesting to observe that blood pigment was entirely lacking, and that also connective tissue was absent from the tumor.

Two Cases of Hebotomy.—SCHAUTA (*Zentralblatt für Gynäkologie*, 1905, No. 20) reports the case of a primipara, aged twenty-three years, with contracted pelvis, mitral insufficiency, and albumin in the urine. The child was presenting in brow presentation; although the pains were good, there was no progress. The patient had been examined before admission to the hospital, and antiseptic precautions had not been strictly observed. The child was living, although the head had not engaged.

In view of the fact that the patient had been examined without rigorous antisepsis, Cæsarean section was declined, and the pelvis was opened on the left of the median line by hebotomy; the forceps was then applied and a living child delivered. Hemorrhage was controlled by tampon, and the wound was closed by drainage. Mother and child made a good recovery, and the pelvis showed good bony union.

He also reports the case of a multipara with contracted pelvis in whom hebotomy was done because the head failed to engage. Delivery was effected by use of forceps, and mother and child recovered well. There was no hemorrhage nor other complications.

Rupture of the Cervix by a Metal Dilator.—BURGER (*Zentralblatt für Gynäkologie*, 1905, No. 19) reports the case of a primipara, aged forty years, in the eighth month of pregnancy, who had eclampsia and had had twenty severe paroxysms. The cervix was 4 cm. long and admitted one finger only; Frommer's dilator was used, and all possible care was exercised in dilating slowly. In fifty-five minutes dilatation had proceeded to No. 7, when it was found that the branches of the instrument had penetrated deeply into the cervix, and that on the left side of the cervix there was occurring free hemorrhage. On removing the instrument it was found that a laceration, between 6 and 7 cm. long, was present at this point; craniotomy was performed, the uterus emptied, and the tear in the cervix closed. The patient died eight hours later from eclampsia. Autopsy showed cerebral hemorrhage, with œdema and hemorrhage in the pleura and liver. The tear in the cervix was found to be 8.5 cm. long and 4 cm. wide. There was no evidence that it opened the peritoneum, as muscular tissue was found throughout the extent of the tear.

Persistent Posterior Rotation of the Chin.—In the *American Journal of Obstetrics* for May, 1905, REED reviews thoroughly this subject, and arrives at the following conclusions:

In labor the face may reach the pelvic floor without anterior rotation of the chin. In most cases, however, anterior rotation does occur spontaneously; when it does not interference is indicated. The child's neck is sufficiently long to permit the head to sink deeply into the pelvis before the thorax is involved. In 22.6 per cent. of cases the chin was delivered without rotation; the maternal mortality of these cases was

practically that of placenta prævia under aseptic conditions, namely, 12 per cent. The fetal mortality is about that of eclampsia under modern methods of treatment.

If the head is not engaged, an effort should be made to produce flexion by the hand, if this fails version should be performed. If the head has engaged, an effort should still be made to produce rotation by the hand. If forceps are used at all, it should be as a last resort before embryotomy. Fetal mortality under the use of forceps is 50 per cent.; only axis traction instruments should be used. If symphyseotomy be elected, it should be performed when the child is vigorous enough to justify the operation.

Puerperal Septic Infection.—The *Practitioner* for March, 1905, is devoted to the discussion of puerperal septic infection. Herman writes upon clinical phenomena, describes the puerperal ulcer, hospital gangrene, spreading traumatic gangrene, sapræmia, septicæmia, pyæmia, peritonitis, late peritonitis, and pelvic cellulitis as different forms of the disease.

Galabin describes the treatment and has found advantage in obtaining material from the uterus for examination in employing the double sterilized tube. The outer and larger tube is closed at the end by a thin india-rubber diaphragm; when this is passed within the cervix the inner and thinner tube is pushed through, it takes up the secretion and is then withdrawn within the rubber.

The uterus is thoroughly irrigated with chinosol, 0.25 per cent.; tincture of iodine, 1 per cent, or lysol, 1 per cent. Vaginal douches of bichloride, 1: 3000 or 1: 4000, are often used. The bowels should be freely opened.

If this does not suffice, the uterus must be thoroughly explored by the finger; retained matter broken down and separated, and then removed by irrigation. If bleeding occurs, packing with iodoform gauze is indicated; suppositories of iodoform, each containing 2 drachms, may be introduced into the uterine cavity daily for some time.

Free feeding is advised, and normal salt solution beneath the skin; fever is to be reduced by cold and quinine and iron may be given; phenacetin for pain, and strychnine for feeble pulse was also recommended. Antistreptococcic serum has been of value in some of his cases.

The author speaks well of collargol, although he apparently has had no experience with that or with nuclein.

So far as operative treatment is concerned, he would limit this to the opening of abscesses; if such are pelvic, they should be opened through the vagina; if abdominal, they should be opened through the abdominal wall. Hysterectomy should very rarely be done, and then if the uterus is large, through the abdomen; and if it is small, through the vagina. Occasionally abdominal section affords a chance for recovery in peritonitis while opening the posterior cul-de-sac is useful in pelvic peritonitis, It is occasionally useful in beginning pelvic peritonitis.

The Prevention of Puerperal Septicæmia.—In the *Practitioner* for March, 1905, HART contributes a paper upon this subject. He lays especial stress upon the sanitary oversight of the patient during pregnancy, and upon the observance of such hygiene that shall bring her into the best possible condition. He urges cleanliness at the time of

labor with thorough disinfection of the hands, and believes that they can be made practically pure for obstetric work without the use of gloves. To one accustomed to operating with gloves there is no objection to their use, although they interfere with the sense of touch at first.

One thorough vaginal examination is usually sufficient and should be made with every precaution for surgical cleanliness. The course of labor should carefully be watched, and forceps should not be used too early nor too late. Deep lacerations should, if possible, be avoided.

He also urges that the placenta should not be delivered too early, and thinks that Credé's method has been harmful in this respect. Laceration of the cervix, giving rise to hemorrhage, should be sutured, and all except the slightest tears of the perineum should be closed. He gives one hot vaginal douche after labor, but no others.

GYNECOLOGY.

UNDER THE CHARGE OF

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Prolapsus Uteri in Virgins and Multiparæ.—ERTZBISCHOFF (*La gynécologie*, August, 1905) states that prolapsus may be found even in newborn infants, in whom there is nearly always an accompanying spina bifida. It is doubtless due to defective innervation of the uterine ligaments and pelvic muscles. In virgins and multiparæ the principal etiological factor is want of muscular tone, due either to vasomotor or atrophic conditions, to which are added repeated and severe bodily efforts.

The treatment varies according to the extent of the prolapsus. If slight it can be corrected by plastic operations on the vagina; a simple prolapsus without accompanying cystocele is best treated by hysteropexy combined with intra-abdominal shortening of the round ligaments. In complete procidentia in a young woman, hysteropexy and plastic operations are indicated, in an old subject supravaginal amputation of the uterus, with fixation of the stump to the abdominal wall, and accompanying colpoperineorrhaphy. All such operations should be supplemented by treatment directed toward improving the muscular tone of the patient

Metritis in Virgins.—DALCHE (*Arch. de thérap.*, 1905, No. 4) believes that infection of the endometrium occurs not infrequently in young girls, due to bad hygiene or accidental blenorrhœal contamination, and is apt to be aggravated under the influence of menstrual congestion. The onset of the disease is more acute than in adults, being marked by severe pains in the hypogastric, ovarian, and sacral regions, accompanied by a *mucopurulent discharge, menorrhagia, and metrorrhagia.* On examination the cervix is found to be hypertrophied and eroded,

the corpus uteri being congested. In a variety described by Pozzi partial or complete arrest of the development of the uterus results.

The treatment consists in those of antiphlogistic remedies, leeching, hot baths, douches, and compresses over the abdomen. Curettement is indicated in cases of menorrhagia due to hyperplasia of the endometrium, dilatation with small laminaria tents in the atrophic variety.

Ovarian Fibromata.—PUECH (*Gazette des hôpitaux*, March 4 and 11, 1905) reviews this subject at length, with especial reference to the diagnosis, which he believes is seldom positively made before the abdomen is opened. A large fibroma of the ovary is apt to be mistaken for one of the uterus, a smaller tumor for a semisolid cyst or diseased adnexa. The mobility of the tumor, the absence of metrorrhagia, the small size of the uterus, and the presence of ascites, all speak in favor of an ovarian fibroid. Dermoid cysts are especially difficult to distinguish from the former. Papilloma is also readily mistaken for fibroma, as well as ectopic gestation.

The writer believes that one cannot affirm that a solid tumor of the ovary is not a sarcoma or carcinoma, but in any case an explorative incision is indicated.

Arterial Tension during the Climacteric.—PAWINSKI (*Gazeta lekarska; Zentralblatt f. Gynäkologie*, 1905, No. 33), as the result of observations in 375 cases, concludes that increased arterial tension is a common symptom of the menopause. It is often observed in women who still menstruate regularly, as well as after the flow has entirely ceased. It diminishes under the influence of profuse flow and nervous influences. This tension is the cause of the tachycardia as cardiac irregularity and so often observed in these subjects.

The climacteric has no particular influence upon the development of arteriosclerosis, though sternocardiac attacks often occur. If aortic lesions are present the heightened arterial tension has an unfavorable influence, but in mitral disease, with low blood pressure, the effect may sometimes be beneficial.

The writer finds that preparations of iodine are not borne well in patients with climacteric arterial tension. He administers ovariin and advises a diet of milk and vegetables.

Combination of Cancer and Fibromyoma of the Uterus.—PIQUAND (*Ann. de gyn. et d'obstétique*, July, 1905) concludes the first half of a paper on this subject as follows: 1. Cancerous degeneration of a fibromyoma is rare, although the writer has been able to collect 45 cases. 2. Usually the fibrous growth is invaded by cancer in the neighboring tissue (especially the endometrium), though exceptionally the original focus may be in the ovary. Cancerous emboli brought through the lymphatics into the tissue of a fibroid from distant organs have been described in only 3 instances. 3. The writer has found 24 authentic cases in which the fibromuscular elements seem to have been actually transformed into epithelial cells (?), though he admits that in some cases pre-existing cells are present in fibroids which may proliferate and develop into a malignant growth. These cells may be derived either from the embryonic remains of the canal of Wolff or Müller, or more frequently are simply epithelial ingrowths into the fibrous tissue

of the endometrium covering the neoplasm, which become isolated, multiply in an irregular manner, and finally develop into primary epithelioma.

Effect of Castration on the Sexual Function.—VINSBERG (*Revue d'obstétrique et de gynécologie*, 1905, No. 7) states that castration does not diminish sexual feeling in women; in fact, it is often increased in women with small, non-developed uteri.

He believes that removal of the uterus and adnexa has little influence of the function; on the contrary, increased passion was noted in several instances. Mandel and Burger, from a series of observations in 25 women, found that in 9 sexual feeling was diminished after castration, and in 6 it was abolished.

[We are more apt to regard the conclusions of the latter writers as more nearer the truth, judging from the extended results of our own practice and observation.—H. C. C.]

DERMATOLOGY.

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Treatment of Mycosis Fungoides by Radiotherapy.—BELOT and BRISSERIE (*Archives d'Electricite medicale*, November 25, 1904) speak of the good results obtained in two cases, one in the premycotic, the other in the fungoid stage. Low vacuum tubes were used (from 4 to 9 H., Holzknecht's chromoradiometer) at each sitting, and at intervals of fifteen or twenty days. Some tumors were removed by 9 H.; others required 35 to 40 H. in all, and sometimes one treatment produced good results. It is not regarded as desirable to cause inflammation. The itching is speedily relieved. The authors are of the opinion that the amount absorbed and the quality of the rays are important factors, but the apparatus employed and the distance of the tube from the skin are of small importance. In one case the treatment extended over a year, the patient increasing in weight during this period. In none of the cases of any disease treated have the authors ever observed any bad effect on the general health of the patient. [Like reports have been made by other observers, but in one case by the abstractor of the above article no benefit was derived by this treatment.]

Glandular and Cutaneous Diseases Healed by Radiotherapy.—R. H. BOGGS (*St. Louis Medical Review*, March 18, 1905) considers it unwise to apply *x*-rays to trivial diseases that can be cured by other less expen-

sive methods. The rays must be prescribed and given in a therapeutic dose for each disease, in order to obtain good results. Tissues are not always affected to the same extent by the same dose, any more than the same dose of a drug will always produce the same physiological effect. Idiosyncrasy is not believed to be a frequent cause of excessive inflammation, but a certain affected area of disease will react more readily than healthy tissue, because the life of the cells is lower. It requires twice as many units of *x*-radiation to produce the same degree of reaction in healthy tissue as it does in many forms of diseased tissue. If the dosage is small, causing stimulation, nutrition is improved, but if large, atrophy and degeneration in one form or another are liable to follow. The author states that 75 per cent. of acne cases can be cured without producing inflammation, but in the other 25 per cent. of cases reaction to the extent of desquamation (as in severe sunburn) is required. He considers that the benefit is due to an induced leukocytosis. [In extolling the *x*-rays for the treatment of this often trivial affection, we are surprised that the author does not caution the operator on the liability to the occurrence of the unpleasant results that may occur from short and infrequent exposures, especially in the form of persistent pigmentations and telangiectases, either of which (when they occur) may be most disfiguring and lasting than the acne.—Abstractor.] Attention is called to the good results in a few cases of Hodgkin's disease and in goitre. A remarkable improvement in the general health usually follows after a few radiations in the former disease, while in the latter the growths are not only reduced in size, but the symptoms of the disease often disappear.

Xanthoerythrodermia Perstans.—H. RADCLIFFE-CROCKER (*British Journal of Dermatology*, April, 1905), under the above provisional title, describes a form of disease which he believes has not been described hitherto. During the past three years he has observed 10 cases of the affection, all of them males, with one exception. Up to the present all the cases have been adults. The eruption begins as pale pink or yellowish patches on the limbs and trunk, the uncovered parts of the body being free or very slightly affected. The patches come out very gradually and in small numbers, being, as a rule, symmetrically placed. As the old patches do not entirely disappear and new ones are continually coming out, large areas are eventually involved. They are usually oval or elongated in shape, and arranged symmetrically on the back in the direction of the ribs, and often but not always in vertical lines on the limbs. Pinching up the skin reveals distinct infiltration, but the patches are not raised above the healthy skin. The surface is smooth on the trunk, but slightly rough or even scaly upon the limbs. The thighs are the parts most frequently first affected, next the legs, then the trunk; the face is nearly always free. In most cases itching is slight or absent. The duration of the disease varies from a few months to many years, and is apparently compatible with perfect health. The affection seems to be fairly amenable to treatment; 2 cases were cured and others improved by the administration of salicin internally and iodine vasogen locally. In the single case occurring in a female exposure to the Roentgen ray cleared up the affected area entirely. Nothing is known as to the etiology. Two of the patients had syphilis, but antisyphilitic treatment pursued vigorously for a year was without effect. Microscopic

examination of excised portions of skin showed absent or atrophied stratum granulosum, absent stratum lucidum, and slight œdema of the epidermis generally. The vessels of the corium were dilated and surrounded by some cellular infiltration. The collagen was apparently normal, with the exception of slight œdema, but there was apparently a reduction in the quantity of elastin, especially in the papillary layer.

OTOLOGY.

UNDER THE CHARGE OF

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Technique of and Indications for Removal of the Malleus and Incus.—HERNRICH NEUMANN (*Archiv f. Ohrenheilkunde*, Band lxiv.), under the above title, discusses the method of anæsthetization of the tympanum and tympanic membrane, the technique of the operation, and the indications for the operation itself. Neumann advises local anæsthesia by the method used in Politzer's clinic for all endotympanal operations. The fluid used for injection beneath the skin of the external auditory canal is a 1 per cent. solution of cocaine, to which tonogen, a preparation of adrenal extract, is added, in the proportion of 1 to 4 drops of tonogen to a cubic centimetre of the cocaine solution. The subcutaneous syringe used for the injection differs from an ordinary syringe, in that the needle is attached to the barrel at an obtuse angle, for ease in manipulation. The needle is introduced beneath the skin of the external auditory canal in its upper part and about 0.5 to 1 cm. outside of the place of juncture of the cartilaginous and bony portion of the canal and is carried in from this point under the periosteum of the bony canal. For satisfactory anæsthesia it is necessary to inject sufficient fluid to elevate the upper and posterior cutaneous lining of the bony canal. The efficacy of this method of cocainizing the tympanum and tympanic membrane is dependent upon the anatomical structure in this situation. The cutaneous lining of the bony canal in its upper part goes over directly into the epidermal layer of the pars flaccida of the tympanic membrane, and, as the pars flaccida has no membrana propria and its inner layer of mucous membrane is a direct continuation of the mucous membrane lining the tympanic cavity, the fluid injected will in part spread under the mucous membrane of this cavity. Sometimes a swelling of the mucous membrane over the promonotory can be observed when this method has been used. The injected fluid also penetrates along bloodvessels which pierce the outer wall of the attic, thus reaching the space of Prussak and the external attic wall. Another

anatomical factor in this location is the strand of connective tissue which is found at the point of union of the os tympanicum and the squamous portion of the temporal bone. This connective tissue is continuous with some of the mucous membrane reduplications of the tympanum. These facts serve to explain the manner in which, in a subperiosteal injection in the bony canal, the fluid reaches the mucous membrane of the tympanic cavity. At least ten minutes must elapse between the time of the injection and the beginning of the operation.

The extraction of the hammer is accomplished in the usual way. After severing the remnant of drum membrane up to either side of the handle of the hammer by means of a round-ended knife, a Delstanche ring knife embracing the handle is passed up as far as possible, severing the tensor tendon and other attachments. The extraction is then completed by means of Sexton's forceps. With these forceps the hammer is seized above the short process and drawn down and then out. After a hearing test the anvil is removed by passing a curette, the curette end of which is bent at an angle of 100 degrees with the handle, into the space previously occupied by the head of the hammer and then sweeping backward and downward with the concavity of the curette directed to enclose the anvil.

In cases where there is found to be purulent inflammation or cholesteatome formation in the attic, Neumann advises the removal of the external attic wall. Two parallel cuts beginning at the inner end 1 cm. long are made through the skin of the upper canal wall and their outer ends connected by a cross-incision. The skin between these incisions is then dissected up and removed. The outer attic wall is then denuded of skin and is removed with small chisels. This removal of the attic wall is quite painless with the local anæsthesia described above.

The indications for the extraction of the hammer and anvil depend on the nature of the suppurative process. Chronic suppuration in the attic when there is no symptom suggesting disease of the temporal bone is an indication for the removal of the ossicles, after a thorough trial of conservative treatment has proved unavailing. Cases of chronic suppurative middle-ear disease with perforation in the anterior segment of the drum membrane are not suitable for this operation, as they are almost never associated with caries of the ossicles. Neumann urges as a point in favor of performing the operation with local anæsthesia that in cases where there is only a suspected involvement of the temporal bone in the carious process (cases which, were the involvement of the temporal bone proven, would be suitable for complete exenteration), the extraction of the ossicles may be done as an exploratory operation without the patient, after undergoing, under general anæsthesia, an operation for removal of the ossicles, refusing, from dread of the anæsthesia, further necessary interference. The extraction of the ossicles, with removal of the outer attic wall, does away with the Stacke operation, since the cases, where extensive interference with the temporal bone is required, are more easily operable by the regular complete exenteration, and cases where this is not required are as satisfactorily treated by the simple removal of the ossicles and the outer attic wall.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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The Function of the Interstitial Gland of the Testicle.—BOUIN and ANCEL (*Journ. de phys. et de pathologie gen.*, 1904, tome vi. pp. 1012 and 1039) publish two contributions on this subject. In the testicles of mammals one may distinguish three types of elements. 1. The seminal cells, which together constitute the seminal gland. 2. The cells of Sertoli, which form a nourishing syncytium for the seminal cells. 3. The large interstitial cells. Recent researches seem to show that these latter cells make up a special gland, and further work, which the authors have confirmed, suggests that at least one function of this interstitial gland is to participate in the nutrition of the seminiferous tubules. A much more important function, however, is the possible relation of the internal secretion from this gland to the sexual instinct of the male.

The cells of the seminal tubules have no general action, at least in animals, for the testicles in cryptorchids of the first order, animals which have normal sexual instincts, were found to contain no seminal cells, while the cells of Sertoli and interstitial cells were well developed. Experimental or pathological stenosis of the vas deferens in animals led in certain cases to complete degeneration and final loss of the seminal cells without, however, loss of sexual instinct. In these animals the cells of Sertoli and interstitial cells were not affected. After removal of one testicle and ligation of the vas deferens of the other the remaining organ showed a compensatory hypertrophy of the interstitial cells, with gradual atrophy and degeneration of the cells of Sertoli. With these changes there is no loss in sexual instinct. The authors believe, therefore, that the sexual instinct is entirely dependent upon an internal secretion elaborated by the interstitial cells of the testicles, the interstitial gland. Dissections upon embryo pigs showed that the growth of the sexual apparatus was dependent upon the development of the interstitial gland, and that the presence or absence of these cells determines the sex of the animal. The female genital gland presents no formation which is homologous to these cells at the same ontogenetic stage.

Finally, arrests in the function of the interstitial gland, demonstrable through lack of growth of the interstitial cells in the testicles of young animals, is accompanied by an arrest in development of the secondary sexual apparatus and the non-appearance of tertiary sexual characters.

The Origin of Acute Miliary Tuberculosis.—SILBERGLEITH (*Virchow Arch.*, 1905, Band clxxix. p. 283) reports the pathological findings from

31 cases of acute miliary tuberculosis, in 23 of which a focus of tuberculosis was carefully searched for in the blood and lymph vessels. In 22 of these cases, or 95.6 per cent., a vascular tubercle was discovered, most often in the pulmonary veins, but also in the thoracic duct, aorta, and other vessels. The author believes that these vascular tubercles of Weigert may be found in all cases of miliary tuberculosis which are carefully studied at autopsy, and explains the failure to localize this process in his 5 per cent. of negative cases principally by the difficulties of technique. The author agrees with Weigert in concluding that for the origin of the typical acute form of generalized miliary tuberculosis a localized tuberculous nodule in a vessel is necessary. It is conspicuous that the vascular tuberculosis arises only exceptionally by the extension of a tuberculous process from the surrounding tissue, but develops usually from the implantation upon the intima of the vessel of a few tubercle bacilli carried there by the blood stream. Occasionally foci of tuberculosis are found in several vessels. From these tubercle bacilli may be swept throughout the body. This fact possibly explains the occurrence of tubercles of different sizes in the same organ and in different organs.

Agglutination in Autoinfection, with Special Reference to Icterus.—Attention has been called by many observers to the fact that serum from certain jaundiced individuals possesses the power of agglutinating the typhoid bacillus.

LUDKE (*Deut. Arch. f. klin. Med.*, 1904, Bd. lxxxi. p. 34) reports the results of a series of observations and experiments bearing upon this point. He examined the blood from 32 cases of jaundice in adults due to various causes, and found that in 28 of this number the serum agglutinated the typhoid bacillus in dilutions of 1 : 20 or over. The agglutination was most constant and took place in the highest dilutions with the serum from cases of catarrhal jaundice. The agglutinating property of the serum withstood a temperature of 55° C. to 60° C. for half an hour, but was destroyed when the temperature was raised to 65° C. to 70° C for an equal length of time. The serum from 7 cases of icterus neonatorum gave no reaction with typhoid bacilli. Further studies showed that the serum from cases of nephritis and diabetes occasionally showed a mild reaction, while with cases of perityphlitis and chlorosis the reaction was frequently present, occurring in dilutions up to 1 : 60. In jaundice the serum agglutinated not only typhoid bacilli, but reacted as well toward bacillus paratyphosus, bacillus coli, bacillus lactis aërogenes, and the spirillum of cholera. Agglutination, however, was never as high or as well marked with these organisms as with bacillus typhosus. The above observations suggested that in jaundice the agglutinating power of the serum was not due to the presence of bile pigments in the blood, but was rather dependent upon a concomitant infection set up by some organism closely allied to bacillus typhosus. Further experiences substantiated this view. Typhoid bacilli were not agglutinated with bile alone, nor with blood mixed with bile. Neither was there any reaction with the serum from animals injected with bile. Taurocholic acid alone had no effect, but the serum from animals injected with this acid showed a slight and inconstant agglutinating property. The serum from animals jaundiced by means of injections of toluylendiamin and inhalations of arseniuretted

hydrogen showed no increase in the agglutinating power for typhoid bacilli; though with ether injections a slight increase was noticeable.

The author concludes that the serum agglutination of typhoid bacillus may take place under three different sets of conditions: 1. After typhoid infection. 2. Following infection by other organisms, as when agglutination takes place with the sera from certain cases of catarrhal jaundice, cholelithiasis, etc. 3. Through a disturbance in the normal relation of the components of the blood, as in certain blood diseases, icterus without infections, etc.

The Disappearance of Bacteria from the Circulating Blood.—The problem of latent bacterial infections has recently received some study, and the work of Tizzoni and Panichi has shown that animals immunized against the pneumococcus may harbor in the circulating blood living pneumococci after subsequent intravenous injections for weeks or even months. SCHWARZ (*Zeitschrift f. Heilkunde*, 1905, Bd. xxvi. p. 293) records some experiments upon the disappearance of typhoid bacilli from the circulating blood of guinea-pigs after intravenous injections of subminimal lethal doses of this organism. He found that the bacteria disappeared from the blood very slowly. After small doses typhoid bacilli could be cultivated from the blood of the animals six and sometimes eight days after injection, although the actual time required for the disappearance of the bacteria from the blood seemed to be dependent upon the amount of the culture injected. The spleen, bone-marrow, and lymph glands harbored living bacteria often in large numbers long after the cultures from the blood gave no growth. These organs almost always contained typhoid bacilli fourteen days after the primary injection, and in one case the bone-marrow was found to contain living bacilli one month after the intravenous inoculation of $\frac{1}{10}$ of an oese of typhoid culture. During the first few hours after injection the numbers of bacilli decrease rather rapidly in the blood and increase proportionately in the spleen. The result of these experiments, the author believes, does not uphold the view that actively bactericidal substances circulate in the blood. The destruction of the injected bacteria may not take place suddenly in the great vessels, but rather gradually in certain organs through the activity of the body cells.

In a paper upon "General Septicæmia," DUVAL and LEWIS (*Journal of Medical Research*, 1905, vol. xiii. p. 534) report among other cases one in which there was a so-called latent infection by bacillus mucosus capsulatus in man. This organism was obtained from the blood of the patient in pure culture a day after admission to the hospital; again after a week; and even in greater numbers, as demonstrated by plate cultures, the day of discharge. On discharge the patient had apparently recovered completely from his illness and looked perfectly well. Two weeks later he was at work

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The first evidence of meningitis appeared two and one-half days before his death; that is, at the same time with the first evidence of endocarditis. On this day there was severe headache and congestion of the conjunctivæ of the left eye. An anxious expression, too, came into the patient's face. The next day the patient was in a semi-coma, being hard to arouse; the neck was stiff; there were movements of

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FIG. 1



Left ventricle, showing vegetations on the aortic valve. Hairs pass through a small and a large perforation.

pneumococci developed in the bouillon, and, after transplantation, on blood serum and in Hiss' inulin medium.

During these last three days the general condition of the patient naturally became worse gradually and progressively, the temperature was most of the time between 105° and 106°, the pulse ran up to 150, and the respirations at one time during the last day were 72.

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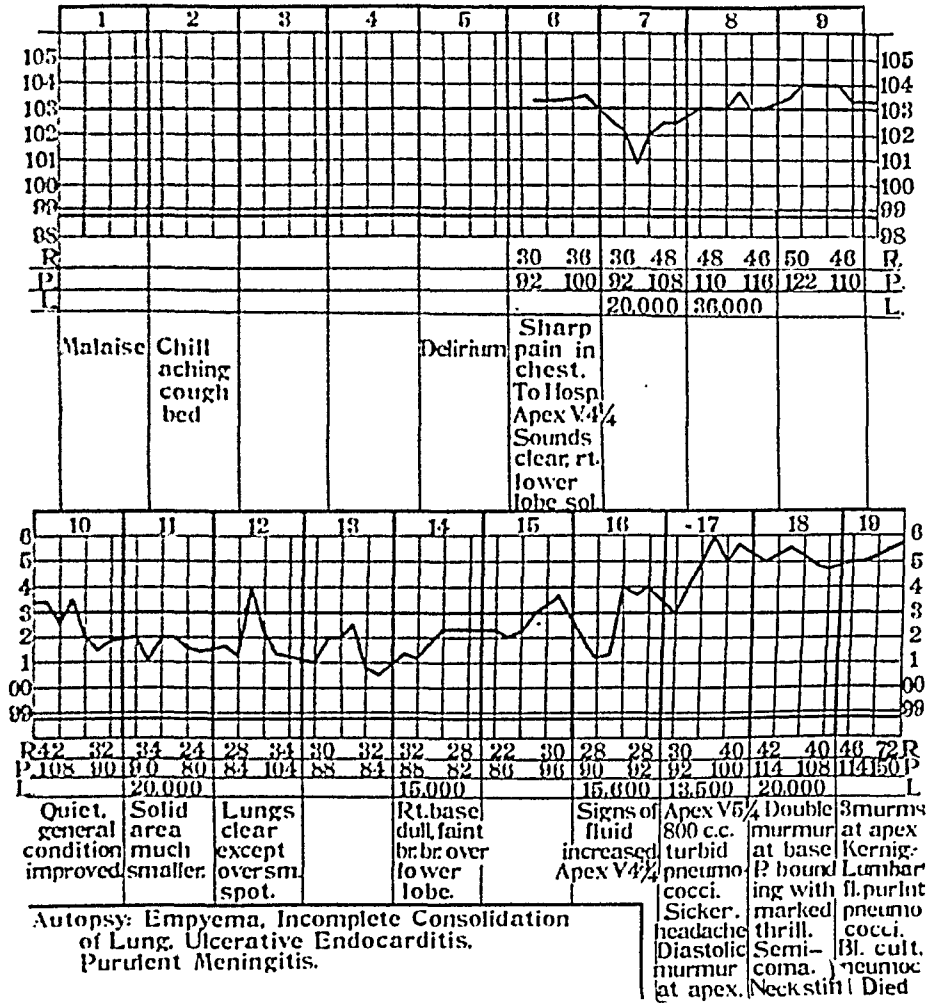
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The leukocytes during his stay in the hospital were counted seven times and ranged between 13,500 and 36,000, the highest number being on the eighth day of his illness, the lowest two and one-half days before death, but on the day before death they rose again to 20,000.

The urine on the seventh day had a specific gravity of 1029, and contained 0.5 per cent. of albumin by bulk and some hyaline,

FIG. 2



Temperature chart with synopsis of symptoms.

granular, and epithelial casts. On the thirteenth day the specific gravity was 1016; no albumin or casts were found. On the sixteenth day the specific gravity was 1015; there was a very faint trace of albumin, but no casts.

Before the onset of this illness the patient is said to have been in good health, though often indulging too freely in alcohol. The

pneumonia, therefore, seems to have come upon him out of a fairly clear sky, and to have run a course not varying greatly from the ordinary except as modified by his alcoholic history, and to have resolved, though slowly and incompletely. When the pneumonia had nearly resolved an empyema developed, and immediately after this a general infection by pneumococci, which attacked the lining of the heart and the membranes of the brain and spinal cord so viciously that within a very few days extreme lesions were produced of ulcerative endocarditis and meningitis, the chief interest of the case arising from the rapidity and the intensity of the effects of the virulent infection by the pneumococci.

The autopsy, made twenty-seven hours postmorten, revealed a resolving pneumonia, an empyema, an exuberant vegetative and ulcerative endocarditis, and an intense meningitis.

The pericardium was normal, the heart muscle showed some grayish mottling. On one of the cusps of the aortic valve was a large, soft, rounded vegetation, one-half inch in diameter with a rough surface and a small perforation through its centre. Just beside it there was another perforation through the cusp. The under surface of the adjacent cusp is a little roughened. The heart weighed thirteen and one-half ounces. The other valves were normal.

The left pleura was normal and the left lung on section showed only a considerable degree of œdema. The right pleura, both visceral and parietal, over the entire posterior surface of the lung, was covered with a layer of shaggy fibrin, and in the pleural cavity was 175 c.c. of purulent fluid. The lung on section was œdematous and congested in the upper lobe, and in the lower lobe dark red, a little mottled, rather granular, tough and flabby, and fragments of it sank in water.

The liver, spleen and pancreas appeared normal.

The left kidney showed one little white infarct at the surface and running in about one inch. Otherwise the kidneys appeared normal excepting some grayness of the cortex on section and congestion of the veins of the surface.

The stomach, intestines, and bladder were normal.

Over the brain and the pia and arachnoid there was an extremely abundant exudate, quite general, including the convexity, but most marked at the central focal portion. There were numerous petechial hemorrhages in the walls of the fourth and lateral ventricles and a few drops of pus in the lateral ventricle.

Ten blood serum tubes were inoculated from the blood of the right and left ventricles and from the pericardium. On one of these inoculated from the left ventricle a growth appeared of pneumococci giving the characteristic reaction with Hiss' inulin medium.

INTRAPLEURAL LIPOMA; ACUTE PERICARDITIS; PERICARDIAL EXPLORATION.

By R. H. Fitz, M.D.,
of Boston.

It is well known that in certain individuals, especially the obese, a large quantity of fat tissue may be found beneath the pleura, and it is easily conceived that a lipoma might be developed from this subpleural fat. Cruveilhier,[1] indeed, mentions the occurrence of fatty tumors of the diaphragm which have forced their way through the fibres of this muscle from the subperitoneal fat, at times accumulated behind the ensiform cartilage. The attached peritoneum is stated to form a true hernial pouch ready to receive the abdominal viscera.

A rounded, lobulated, subpleural lipoma somewhat larger than a plover's egg was found by Clark[2] in the right pleural cavity of a woman, aged sixty-five years. Its "base was spread out between the muscle fibres of the diaphragm like the mycelium of a mushroom," and the tumor resembled "at first sight, a diaphragmatic omental hernia." He calls attention to a normal "outgrowth of fat from the diaphragm situated on the right side anteriorly close to the reflection of the pericardial pleura on to the diaphragm. This may, in cases of general obesity, become occasionally so increased in amount as almost to assume the appearance of a fatty tumor, but does not, of course, constitute a true tumor, since it is but part of a general physiological process."

Conner[3] records a somewhat similar observation. He found in an elderly woman who died of cerebral hemorrhage, a sessile, fatty tumor measuring 4 x 3 cm. It was covered by pleura and projected from the diaphragm into the left pleural cavity a little to the left of the apex of the heart. At its base there appeared to be a deficiency in the diaphragm, which was offered as a possible explanation of the origin of the tumor.

The subpleural fat along the intercostal spaces is often abundant, and Rokitansky[4] states that, at times, it projects into the pleural cavity as notable, lobulated masses.

Lancereaux[5] knows of but one well-authenticated instance of a subpleural lipoma. Chiari[6] is stated to have observed in an elderly

[1] Traité d'anat. path. gén., 1856, tome iii. p. 310.
[2] Transactions of the Pathological Society of London, 1887, vol. xxxviii. p. 324.
[3] Proceedings of the New York Pathological Society, 1897-98, p. 43.
[4] Lehrb. d. path. Anat., 1861, Bd. iii. p. 39.
[5] Traité d'anat. path., 1879-81, tome ii. p. 147.
[6] Gussenbauer, Arch. f. klin. Chir., 1892, Bd. xliii., Festschr. 233.

female a pendulous lipoma of the size of a walnut projecting into the pleural cavity from the seventh left rib.

The subpleural lipoma of the costal wall may become so large as to be of practical importance, as illustrated by the experience of Czerny.[1] This surgeon removed a lipoma of the size and shape of a man's head from below the left shoulder-blade of a male patient, eighteen years old. The tumor was of rapid growth and was found at the operation to be continued into the thorax between the seventh and eighth ribs, which were separated by a gap of the size of a hen's egg. The patient died of septic pleurisy, and the subpleural cavity within the chest from which the innermost portion of the tumor was removed, contained several sinuses. The growth was considered to have proceeded from the endothoracic fascia beneath the pleura. Several years later a like experience was published by Plettner[2] as having been met by Volkmann in the practice of another surgeon. An elderly woman had a lipoma of the size of a child's head projecting from the left wall of the thorax. At the operation it was found to be prolonged into the chest by a pedicle between the fourth and fifth ribs in the axillary line. The outer portion of the tumor was removed, but the patient died of septic pleurisy and pericarditis due to an ichorous inflammation of the pedicle. At the autopsy the latter was found to be continued into a flattened subpleural lipoma as large as a hen's egg. Gussenbauer[3] examined a female patient, aged twenty-nine years, who had a tumor at the upper border of the left mammary gland. It was of the size of a nut and lay in the second intercostal space between the edge of the sternum and mammillary line, beneath the mammary gland and the pectoral muscles. The latter structures could be moved over the tumor which could not be displaced from the thoracic wall. Three years later the patient again was seen, the tumor having become enlarged in the mean time, and especially within the previous months. It was regarded as an encapsulated lipoma proceeding from the second intercostal space, on account of its mobility, consistency, lobulated surface and place of fixation. Its matrix was considered to be the subpleural fat tissue, as in the case reported by Czerny, which Gussenbauer also had seen, and in accordance with the previously mentioned statement of Rokitansky concerning the subpleural fat. At the operation a pedicle twice as thick as the finger was extended from the tumor into the chest through the widened second intercostal space. Traction upon the pedicle brought to view a portion of the intrathoracic tumor. The latter was enucleated by the finger from its subpleural attachments and was removed. It was a little smaller than the outer tumor, about the size of a man's fist, and was contained within a

[1] Wien med Woch., 1875, Bd xxv p 166
[2] Inaug Diss., Halle, 1889
[3] Loc cit., p 322

subpleural cavity. The entire tumor was twice as large as the fist, and weighed over five hundred grams. The patient recovered.

The fat tissue of the mediastinum also is known to have served as the place of origin of intrathoracic lipomata which have projected externally from the wall of the chest, and have been submitted to surgical treatment. Cruveilhier[1] states that during an operation for the removal of a lipoma in front of the sternum the surgeon found several prolongations continued from it along the edges of this bone into the anterior mediastinum. Most of these were withdrawn and cut off. The patient died of suppuration of the anterior mediastinum. He mentions[2] also that Morel-Lavallée successfully removed from the sternal region the superficial portion of a lipoma which was continued into the anterior mediastinum by a prolongation which was not disturbed.

Kronlein[3] reports the case of a female infant, aged one year, operated upon by Langenbeck. At the age of six months a small tumor near the right breast was noticed by the parents. It grew rapidly, and at the time of operation formed a lobulated mass extending from the right axillary line to 3 cm. beyond the left sternal edge, and from two fingers' breadth below the right clavicle to the xiphoid cartilage. At the left of the tumor there was dulness on percussion preventing an accurate determination of the outlines of the heart. The apex beat was palpable in the sixth interspace, 2 cm. outside the mammillary line. The tumor increased considerably in size in the course of five days, and Langenbeck suggested its probable lipomatous character and congenital origin from the anterior mediastinum with subsequent perforation of the thoracic wall. On exposure of the tumor it was found to be continued into the chest through the third intercostal space, 1 cm. from the right sternal edge, by a pedicle as thick as the thumb. The pedicle was cut across at the level of the ribs, and the external tumor removed. The infant died of erysipelas. At the autopsy a rounded lipoma as large as a child's head, and covered with a firm, fibrous capsule, was found completely filling the anterior mediastinum, and continuous with the pedicle above mentioned. It projected 2 to 3 cm. beyond the right sternal edge, and filled almost the entire anterolateral portion of the left thoracic cavity, leaving only a small space below and behind, in which lay the compressed lung and the displaced heart.

The following unique case shows that subpleural lipomata may have a medical as well as a surgical interest. The patient, a fisherman, aged thirty-four years, lean, always well with the exception of a "typhopneumonia," entered the Massachusetts General Hospital January 4, 1905. He had been seized with a severe chill ten

[1] Loc. cit., p. 315. [2] Loc. cit., p. 322.
[3] Arch. f. klin. Chir., 1877, Bd. xxi.; Supplement, 157.

days previously, followed by pain across the chest aggravated on deep inspiration. There was a distressing cough, with rusty sputum and considerable dyspnœa. The physical signs indicated consolidation of the left upper lobe.

In the lower left axilla, corresponding to the region of the inferior third of the lower lobe of the lung, there were dulness on percussion and absence of respiratory and vocal sounds. The midaxillary region was occupied by a wedge of normal resonance and respiratory sounds, with its apex in the vicinity of the left nipple. There were dulness, diminished fremitus, and feeble vocal and respiratory sounds in the left back between the angle of the scapula and the spine. Numerous medium, moist rales were heard at the base of the left lung behind. The left border of cardiac dulness was four and three-quarter inches from the median line, and the right border of dulness was at the right sternal edge. The apex beat was audible in the fifth intercostal space four and one-half inches to the left of the median line. The heart sounds were regular, rapid, and distant; the second sound was strong at the apex. Adventitious sounds were not heard. There were 41,200 leukocytes per cubic millimetre and 80 per cent. of hæmoglobin.

During the subsequent four days the temperature fell from 104° to 101°, near which latter point it persisted. In the same period of time the pulse fell from 145 to 100, near which rate it subsequently remained. The respiration at first was from 50 to 35, but after five days ranged from 25 to 30, until within two days of his death, when it rapidly rose to 45. Death occurred two weeks after entering the hospital.

For a few days he was mildly delirious, the white count remained high, the signs of consolidation persisted, but on the fifth day the failure to improve and the faintness of the heart sounds suggested the possibility of an acute pericarditis.

The general condition of the patient then became somewhat better, but the physical signs persisted and the leukocyte count remained high. Attention was constantly directed to the pericardium, as well as to the lungs and pleuræ, in search of an explanation of the persistent elevation of temperature, and the continued leukocytosis.

Ten days after entrance and the twentieth day of the disease it was noted that the signs of consolidation continued in the left lung. The right border of cardiac dulness remained at the right sternal edge, the cardiohepatic angle was obliterated, and the heart sounds were faintly heard. The pulse became paradoxical. Although dulness persisted in the left back between the angle of the scapula and the spinal column, the vocal resonance and fremitus and the respiratory sounds were normal.

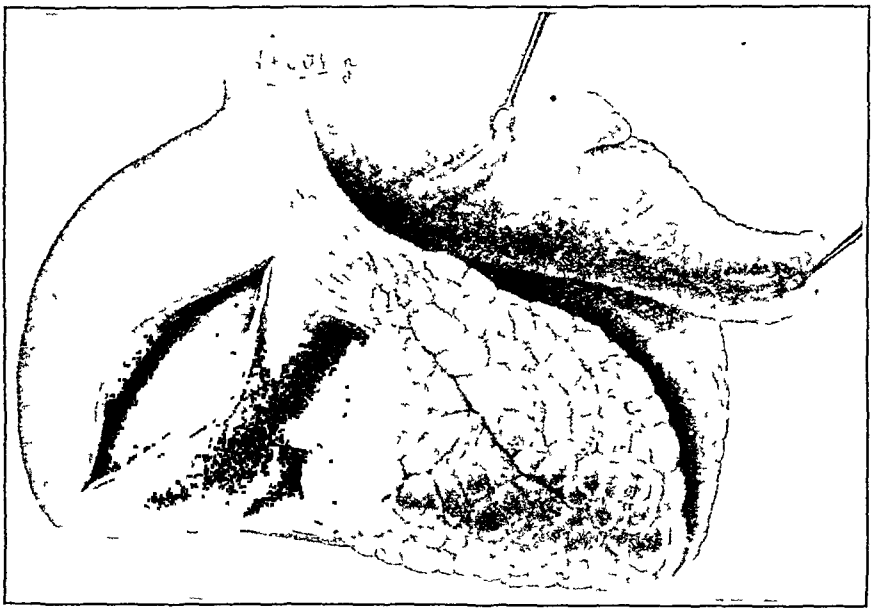
On the following day, the twenty-first of the disease, the pericardium was tapped in the fifth interspace near the left border of the sternum, and again in the left fourth interspace some two inches

from the sternal edge. Although the cannula entered at least an inch and a half in each place, and the surface of the heart was reached, there was no escape of fluid. Two days later, in consequence of attacks of dyspnœa and cyanosis, although there was no change in the physical examination of the chest, the exploratory trocar and cannula were inserted through the sixth left interspace in the axillary line into the subjacent area of dulness with the thought that pus might be present. The instrument entered readily between two and three inches and the tip was moved about freely. After withdrawal of the trocar no fluid escaped, nor did air enter the cannula. The trocar was reinserted and withdrawn and a second attempt at aspiration was made, but without effect. When the cannula was removed it was found to contain a fragment of soft material suggesting fibrin, but which, on microscopic examination, proved to be fat tissue.

My assistant, Dr. Beals, was asked to explore the pericardial cavity from the left xiphicostal angle after the patient's death, which took place the same day. He withdrew about a pint of creamy pus.

A postmortem examination was made by Dr. James H. Wright, from whose record the following abstracts are made: "On opening the pericardium a large amount, probably 500 c.c., of yellowish opaque fluid gushes forth. . . . Adherent to the inner aspect of the pericardial sac and to the surface of the heart everywhere is a dirty yellowish, finely shaggy or rough fibrinous layer 1 to 3 mm. thick. . . . The epicardial fat over the right ventricle near the pulmonary artery is much larger in amount than normal, forming a layer 3 or 4 mm. thick. . . . Attached to the pericardium on the left side and apparently continuous with the fat tissue of the superior mediastinum is a large mass of fat tissue of about the volume of a newborn child's head, divided by delicate connective-tissue septa into larger and smaller lobules. It evidently occupied the inferior and anterior portions of the left pleural cavity, the left lung being behind and above it. The inferior lobe is of about one-third the normal volume. . . . On section it is dark red in color, moist and not obviously atelectatic. The appearance of this lobe of lung suggests rather a hypoplasia than atrophy and atelectasis. The large amount of fat tissue just referred to is more or less adherent by fibrous adhesions to the diaphragm, to the pericardium, to the parietal pleura, and to the inferior lobe of the left lung. Portions of it were left in the body on removal of the thoracic viscera. As far as could be determined the shape of the mass of fat tissue was more or less that of a pear, the small extremity being attached most firmly to the external surface of the pericardial sac along a line extending from the anterior mediastinum in the region of the pulmonary artery (where it seemed to be continuous with the fat tissue of the pericardium) downward on the left side toward the posterior mediastinum. The

mass of fat tissue appeared to be covered in places by a thin, shiny layer resembling the pleura. The superior lobe of the left lung is voluminous and extremely resistant to the touch. On section the cut surface is grayish, granular, firm, and when scraped with a knife yields a rather thin, reddish fluid. The pleural surface of the superior lobe is grayish and opaque, not shiny. . . . Fluid of pericardium aspirated postmortem shows growth of pneumococci by culture. . . . Most of the air cells in the section from the superior lobe of the left lung contain an exudate consisting chiefly of fibrin in dense strands and masses. In a few places there is an outgrowth of fibrous tissue into the air cells, not sufficiently extensive to justify calling the condition organizing pneumonia.



Intrapleural lipoma proceeding from the fat tissue of the superior mediastinum

The appearances in the section indicate a lobar pneumonia in which resolution has not been thoroughly established."

It is evident from the history of the patient and from Dr. Wright's examination that a pneumococcal pericarditis developed without characteristic symptoms or signs in the course of an acute pneumonia of the left upper lobe and that the resolution of the latter was delayed. Owing to the persistent solidification of this lobe and the presence of a large intrapleural lipoma of mediastinal origin the pericardial exudation was not enabled to cause a lateral increase of pericardial dulness. On the contrary, as was made evident by aspiration, the fluid was accumulated behind and below the heart.

The congenital origin of the lipoma is indicated by the accompanying hypoplasia of the lower lobe of the left lung. Its discovery

was wholly unexpected, although the physical signs and the result of aspiration would permit a diagnosis in a second case. Its significance to the physician lies in its presence being a satisfactory explanation for an unusual distribution of pericardial exudation, and its localization offers an opportunity of error in the diagnosis of a circumscribed pleuritic exudation.

It is a matter of common knowledge that even considerable quantities of liquid pericardial exudation occasionally escape recognition. Characteristic symptoms may lack and the heart adapt itself to the encumbrance of moderate accumulations of fluid, and, as a rule, the quantity present is small. Pericardial paracentesis, therefore, is of relatively infrequent occurrence. During a period of thirty-four years at the Massachusetts General Hospital there are noted in the index of the records 150 cases of acute pericarditis, of which 14 were tapped; 5 of these died, 4 were discharged much relieved, 2 relieved, and 3 well. In 4 cases no fluid was obtained, while in the remaining 8 quantities varying from one drachm to one quart were removed. In 6 cases less than four ounces were obtained.

Since October, 1896, evidence of acute pericarditis has been noted in twelve autopsies. The largest quantity of fluid found was nearly one pint, and in the patient concerned the existence of a pericarditis was not suggested until the day of death. In a second instance a half-pint was present, but in the majority of the cases the quantity of fluid observed was less than five ounces.

The diagnosis of an exudative pericarditis is determined by rational signs indicative of an infectious process and by physical signs calling attention to the seat and extent of the exudation. The rational signs alone are not to be depended upon to direct suspicion to the pericardium, since pericarditis usually is secondary to an inflammatory process elsewhere, for example, in the lung, pleura, or peritoneum, the symptoms of which are likely to mask those proceeding from the pericardium.

Especially significant in the etiology of the cases tapped in the hospital were acute pneumonia and rheumatism, each in four cases, while the pericarditis was regarded as primary in three instances. The diagnosis is comparatively easy when the course of the disease can be traced from the initial pain and friction sound to the gradually increasing area of cardiac dulness of characteristic outline, with fading rub and increasing remoteness of cardiac sounds. It is rendered more difficult when the adherent lungs prevent the lateral distention of the pericardial sac, the adjacent lung is solidified, the pleural cavity contains an exudation or the anterior wall of the heart is united to the parietal pericardium by old or recent adhesions. To these familiar obstacles a new source of difficulty is to be added—viz., the intrapleural lipoma, especially when it attains the size and position shown in the case here reported, as well as in that of Krönlein-Langenbeck.

The experience above recorded indicates the essentially exploratory nature of pericardial paracentesis and emphasizes the importance of a thorough exploration in doubtful cases. As usually performed, the needle is inserted into the fourth or fifth left intercostal space near the sternal edge or some two inches from it. The failure to obtain fluid at these points in the case here reported led to the assumption that the exudation was largely fibrinous. The absence of an increased area of dulness at the right of the sternum negatived the thought of tapping at Rotch's point in the fifth right interspace, one and one-half inches from the sternum. The positive result of the postmortem puncture in the left xiphicostal angle makes it obvious that this way into the pericardium should always be favored in cases of suspected exudation, if not in the first instance, certainly when aspiration at other points has proven ineffectual.

Larrey[1] originally recommended this route for pericardial drainage, and gave specific directions for performing the operation. He was led so to do from having successfully drained in this region a stab wound, which gave satisfactory evidence of communicating with the pericardial cavity. Roberts,[2] however, objected to paracentesis in this situation unless the case were exceptional, from the possibility of wounding the diaphragm and liver, and from the difficulty of determining the edge of the ensiform cartilage in fat persons. In two cases in which I have punctured the pericardium in the left xiphicostal angle, although fluid was not obtained, no ill effect followed, and the condition of the patients improved temporarily at least, after the exploration was made.

From the facts above narrated, and from those reported by F. C. Shattuck[3] and others, an exploratory incision in the xiphicostal region seems advisable in urgent cases when exploratory puncture has afforded no relief. Roberts[4] recommended exploratory incision as far as the pericardium when there was a doubt between a large pericardial exudation and a dilated heart. More recently he states[5] that incision is better than aspiration, even when the affection is not supposed to be purulent. Brentano[6] advocates in all cases pericardial incision with previous costal resection as less dangerous than paracentesis, and Weintraud[7] prefers incision to paracentesis. Although Roberts[8] now is in favor of exploratory aspiration in the upper part of the left xiphoid fossa, he considers that this region does not afford room for incision and drainage. Ogle and Allingham,[9] on the contrary, recommended the plan

[1] Clin. Chir., 1829, tome ii. p. 303. [2] Paracentesis of Pericardium, 1880, p 66.
[3] Transactions of the Association of American Physicians, 1897, vol. xii. pp. 193, 196.
[4] Annals of Anatomy and Surgery, 1881, vol. iv. p. 250.
[5] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1897, vol. cxiv. p. 649.
[6] Deutsch. med. Woch., 1898, Bd xxxii. p. 507.
[7] Lehrb d. klin. Untersuch. Meth., 1905, Bd. ii. p. 380.
[8] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1897, vol. cxiv. p 652.
[9] Lancet, 1900, vol. i. p. 693

of reaching the pericardium originally outlined by Larrey, followed by the removal of two inches of the seventh left costal cartilage, if necessary. Latham and Pendlebury[1] reported the successful performance of this operation with a vertical instead of an oblique incision and the removal also of two inches of the sixth costal cartilage, and Pendlebury[2] has operated upon two additional cases, one of which recovered.

Mintz[3] also has successfully drained the pericardium in the region recommended by Larrey.

There is, therefore, no doubt that aspiration through the left xiphicostal space has disclosed pericardial exudation which was not made evident by puncture elsewhere, and that successful drainage has been established through the same region. Consequently this route is to be recommended for exploratory aspiration, incision and drainage, since it gives ample room (by resection of the costal cartilage, if necessary), is free from risk to the pleura and peritoneum, and enables injury of the internal mammary vessels to be avoided or controlled. In critical cases when aspiration causes no outflow of fluid and the tip of the instrument can be moved about within the pericardium exploratory incision should speedily follow.

THE TREATMENT OF CHRONIC OBSTRUCTION IN THE LARYNX AND TRACHEA.[4]

By John Rogers, M.D.,
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The causes of chronic obstruction in the larynx and upper trachea are generally classified as follows:

1. Cicatrices of traumatic, operative, or pathological origin. When following tracheotomy the deformities may be due to (*a*) contraction of the posterior soft part of the trachea following separation of the anterior segments by the cannula; (*b*) dislocation of the cricoid forward and downward, especially when the fistula is through or close to it; (*c*) overriding of the cut anterior ends of the tracheal cartilages in too long a fistula; (*d*) inversion of the cut anterior ends of the tracheal rings, especially if the fistula is to one side of the median line; (*e*) contraction of the trachea around the cannula due to sloughing of the cartilages or rings; (*f*) spur formation posteriorly (as after enterostomy), especially in combination with contraction of the posterior soft portion of the trachea, or dis-

[1] Lancet, 1903, vol. i. p. 798.
[2] Ibid., 1904, vol. ii. p. 1145.
[3] Centralbl. f. Chir., 1904, Bd. xxxi. p. 59.
[4] Read before the American Laryngological Association, Atlantic City, June 8, 1905.

location of the lower segment forward and the upper backward, or both.

2. Granulations around a cannula or intubation tube which are much less common than is generally believed. They have been described as occurring at the upper anterior angle of the fistula for a tracheal cannula, or opposite the usual fenestrum on the posterior tracheal wall, or at the lower end of the cannula or around its whole length, or about the upper or lower end of an intubation tube.

3. Hypertrophic subglottic laryngitis most commonly encountered in retained tube cases, less often in those with a retained tracheal cannula and almost always after diphtheria.

4. Prolonged and repeated diphtheritic inflammation, which must be exceedingly rare in these days of antitoxin, although it is worth noting that there is at least one case on record in which the exudate was demonstrable on the 151st day.

5. Postdiphtheritic paralysis of the laryngeal abductors, which is sometimes described and must be very rare, but clinically is indistinguishable from the following:

6. Atrophy of the laryngeal abductors from disuse in retained tube and tracheotomy cases.

7. Obturation or compression of the respiratory tract by neoplasms is common and involves first the removal of the tumor and next the treatment of any concomitant or resulting cicatricial stenosis.

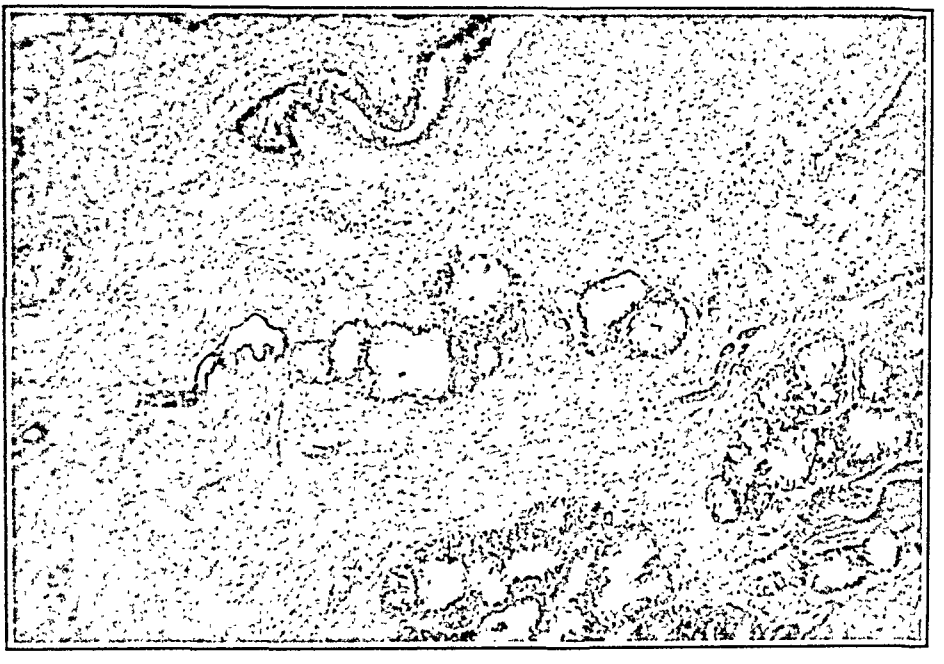
Of these "chronic hypertrophic subglottic laryngitis" needs some discussion. It was described by Kohl[1] as one of the common causes for retention of the tracheal cannula after acute diphtheritic stenosis long before the days of intubation, but at present is generally regarded with suspicion or mistaken for "granulations" or a cicatrix. I had the correct diagnosis first forced upon me by undertaking laryngotomies to remove these supposed granulations or cicatrices in cases of retained tubes. The gross appearance was that of a general velvety thickening of all the soft parts within the thyroid and cricoid cartilages, which bled easily, though there was no ulceration. The vocal cords and ventricles showed only as a slight tumefaction or dimple, and when the divided thyroid alæ were brought in apposition the hypertrophied mucous membrane completely occluded the laryngeal lumen. The futility of operation in such a condition is obvious and to elucidate its pathology, as clinically at least, it is one of the commonest causes of retained intubation tubes, I must repeat in full the findings I have been able to collect.

I am indebted to Dr. F. Tilden Brown for the first specimen, that of a child intubated at the Willard Parker Hospital June 28, 1896. Autoextubation, followed by closure of the larynx within a

[1] Archiv f. klin. Chir., 1887, Band xxxv. pp. 75 und 403

few minutes, necessitated tracheotomy about a month later. A year subsequently, or on July 31, 1897, as the stenosis was unchanged, Dr. Brown inspected the larynx through a thyrotomy at the Presbyterian Hospital. There was an even thickening of the soft parts throughout the whole interior of the laryngeal cartilages. There was no sodden or watery appearance such as might be produced by œdema, but the thickened tissue seemed firm and vascular. Bleeding was easily excited by sponging. There was no sign of ulceration nor cicatrization, and the soft parts were apparently covered everywhere with epithelium. A vertical strip of tissue was then cut from the whole thickness of the soft parts on each side of the inner surface of the larynx. The pathologist of the hospital reported that

FIG. 1



these strips showed "infiltration with many small round cells, in places apparently granulation tissue. Over some thickened areas there is thin mucous membrane." It would be difficult to cut sections of such strips in a plane which would demonstrate the presence and integrity of the epithelium throughout, in other words, to prove by the microscope the absence of ulceration. But this may be inferred from the gross findings in spite of the use of the words "granulation tissue," which are intended to indicate a state of exudative and productive inflammation. This child died later of pneumonia.

The operation did not succeed in making respiration possible without the tracheal cannula.

I am indebted for the second case to Dr. Dillon Brown. This

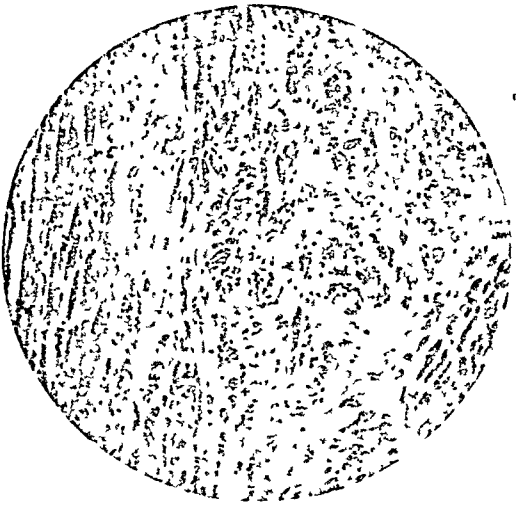
child was intubated by him for laryngeal diphtheria in the first week of February, 1897. The disease subsided, but the stenosis

FIG 2



persisted and necessitated "retention" of the tube. Respiration without it was barely possible for about an hour, but no longer, and finally after autoextubation at the end of a month from the time of

FIG. 3

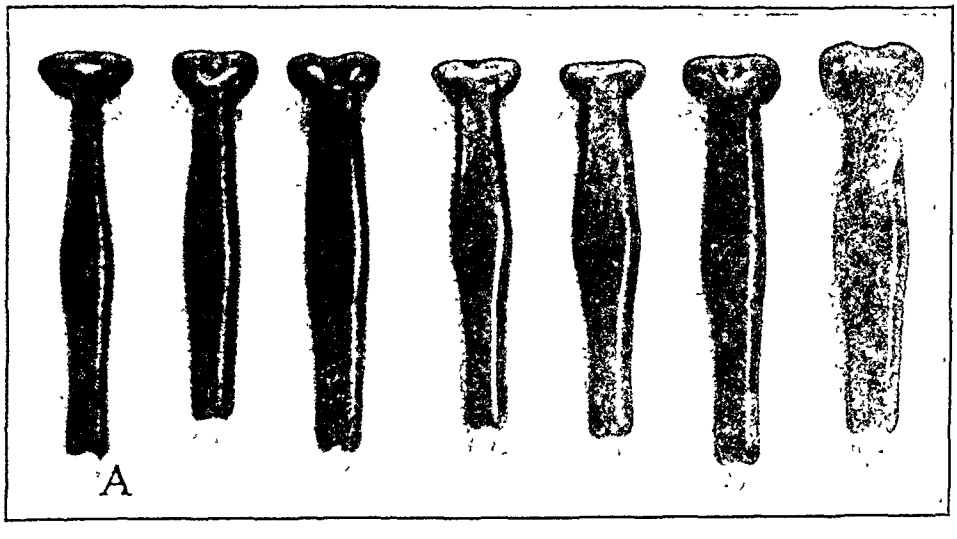


first insertion asphyxia occurred. At autopsy the intralaryngeal soft parts were found to be evenly thickened to such an extent that

the lateral portions of the larynx were in contact in the median line. The tissues were moderately soft and compressible enough to permit the introduction without undue force of the tube ordinarily employed for a child of this age and size.

Dr. W. N. Berkeley, the pathologist then in Dr. Brown's laboratory, cut some sections for me transversely through the centre of the larynx and made the following report: "The mucous membrane, submucosa, and remaining soft parts within the larynx appear much swollen and thickened by a growth of new cells. The vessels are numerous and congested. There is an irregular increase in the superficial epithelium and an hypertrophy of the subepithelial papillæ. There is no evidence of ulceration with loss of substance. The striated muscle fibres, both in front and behind the arytenoid cartilages, are present in the usual amount, but like all the other soft tissues, are infiltrated with columns of small round cells. In all probability this infiltration would interfere with or abolish the function of the muscles. The cartilage is normal."

FIG. 4



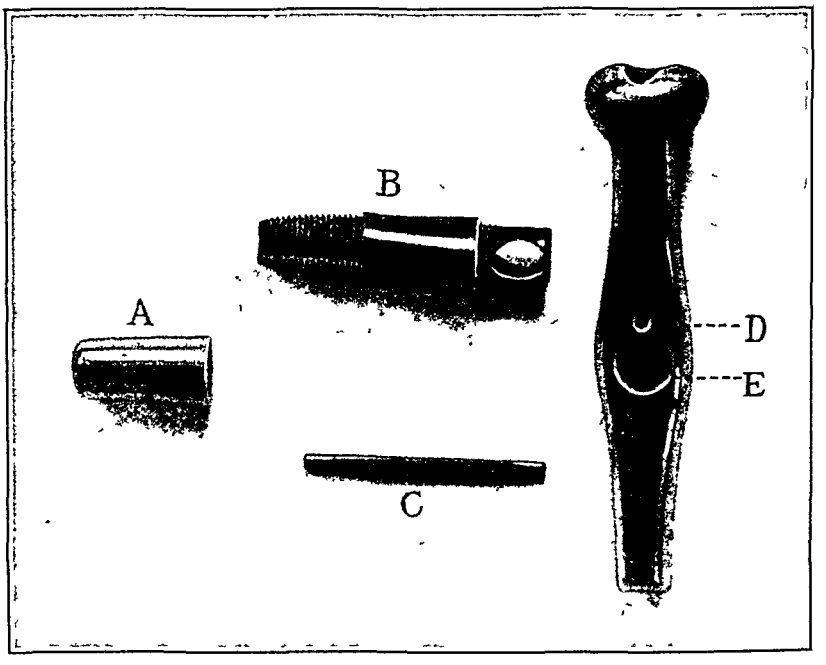
The third specimen is from Case VI. and is one approaching a cure. This child had worn an intubation tube for nearly three years and had never been tracheotomized. Only during the latter two months of tubage (and her life) was natural respiration possible for even a few hours.

The gross appearance was one of smooth, velvety thickening of all the soft parts within the laryngeal cartilages. This thickening reduced the lumen of the larynx to about one-third of its natural size. Within the anterior portion of the cricoid there was an irregularly circular area about one-quarter of an inch in diameter, which was evidently a cicatrix resulting from an ulcer, and another similar area an inch farther down and opposite the lower end of the tube

in the anterior and lateral portions of the tracheal mucous membrane about one-half inch in diameter. These areas did not constrict the lumen of the larynx or trachea, but, on the contrary, seemed slightly depressed below the surrounding soft tissue. Ulceration had therefore occurred at these points and the healing had increased rather than decreased the lumen of the passage.

To make clear the explanation of the treatment proposed I must anticipate by stating that by a "normal" tube is meant one of the ordinary O'Dwyer instruments as originally made by George Ermold & Co. in seven sizes, usually distinguished as suitable respectively for a child one year old or younger—two, three, four to five, six to seven, eight to nine, and ten to twelve years of age. For adults the

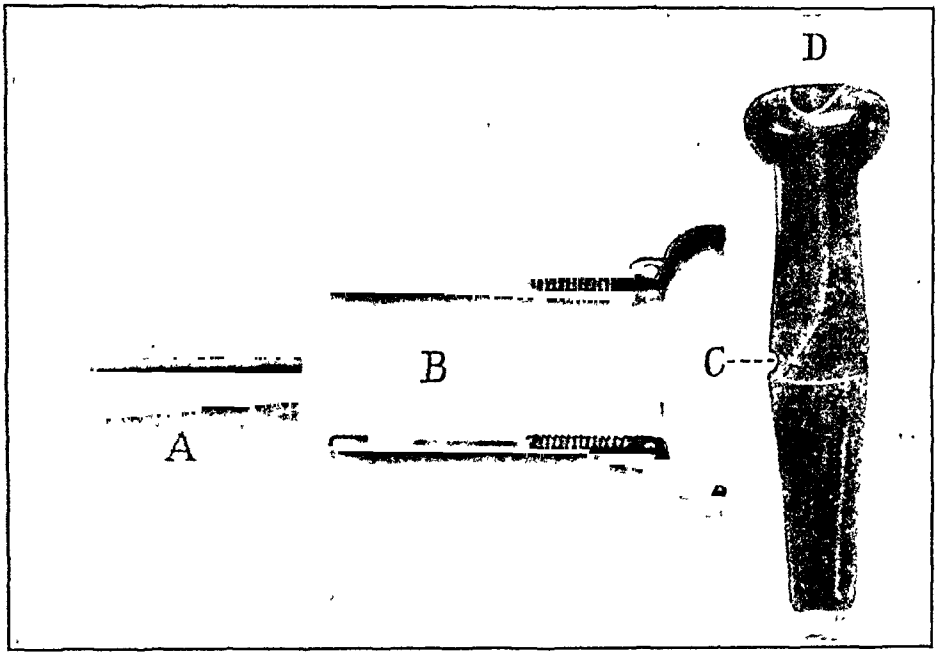
FIG. 5



sizes run from one to ten inclusive. By "special" tube is meant one of the same length as the normal, but in which the neck or constricted portion just below the head or the part below the retaining swell is greater in the transverse diameter generally by $\frac{1}{32}$ inch or some multiple of this amount. The "retaining swell" or bulging central portion of the tube must at the same time have a transverse diameter at least $\frac{3}{32}$ inch greater than the neck or the tube will certainly be coughed out. In cases of hypertrophic laryngitis which are totally unable to breathe without a tube this difference is too slight and a difference of $\frac{4}{32}$ or even $\frac{5}{32}$ inch may be required. Life will often depend on this $\frac{1}{32}$ inch.

By a "plugged" tube is meant one fitted with a plug made to screw accurately into a perforation in the anterior wall of a normal or special tube through a tracheal fistula. The plug is perforated at its inner end by a hole which exactly corresponds in size with the lumen of the tube. The plug is provided with a retaining pin, which, when the plug is in place, prevents it from rotating and so obstructing in the slightest way the lumen of the tube. By previous measurement the tube has a hole bored in its anterior wall, which when the tube is inserted lies opposite the tracheal fistula. Into this hole the plug is then screwed and "pinned" at right angles to the tube. A cap is then screwed down over the outer end of the plug

FIG. 6

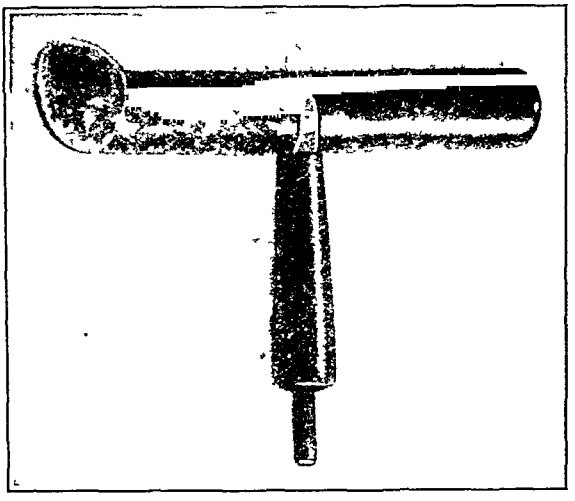


to hold the pin in place. By a "clamped" tube is meant one fitted with a detachable pair of metal clamps made on the principle of an obstetric forceps. In the sides of the tube are cut grooves, which, when the instrument is in place, lie at the level of the tracheal fistula. Through this the clamps are made to grasp the tube and are then held in place by a collar screwed down around them until the collar touches the tube. The plugged or clamped tube is a guarantee against autoextubation and at the same time keeps the tracheal fistula from closing.

The statistics of the 22 histories which could be submitted in full, did space permit, include 3 deaths, one from nephritis and pneumonia, 1 from autoextubation and asphyxia, 1 from asphyxia due to the clogging of a metal tube; 2 failures to cure; 7 still under observation. I do not consider a case cured until at least a year has

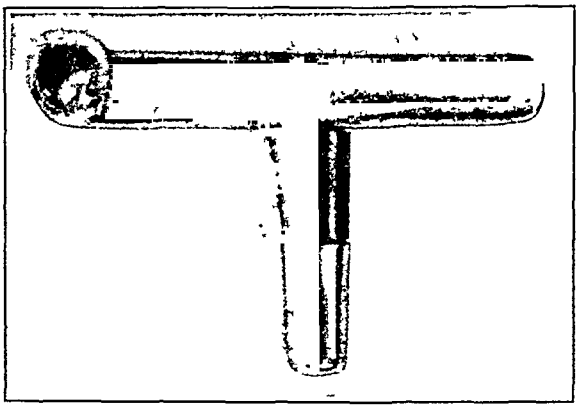
elapsed after the last intubation without the appearance of any signs of stenosis. Of these 7 cases: 3 are able to breathe naturally for a few hours or days, although the obstruction when first seen was complete; 2 (adults) have been from one to three weeks

FIG. 7



without a tube, which was only reinserted when the laryngoscope showed evidence of beginning recontraction (these 5 are therefore still wearing tubes); 2 apparently cured, but the requisite

FIG 8



year has not yet passed; 10 cured, that is, they have remained free from any obstruction for at least one year and some for four or five years. All of these have voices which range from perfect to hoarse.

CASE VI.[1]—*Stenosis due to hypertrophic laryngitis following intubation persisting unchanged for two and a half years; some improvement during the next six months with "special" rubber tubes; adductor spasm evident; more marked improvement with special metal tubes; death ultimately from nephritis and pneumonia.*

G. S., aged three years, was intubated for diphtheria at the Willard Parker Hospital in July, 1902. The stenosis persisted and the tube was worn continuously thereafter with frequent changes until February, 1904. On this date a special rubber tube, of the four to five length, was inserted, having the neck and retaining swell one-thirty-second of an inch larger in diameter than the normal measurement. During the change absolutely no air passed through the larynx, no bleeding was produced, and no force was required for intubation. The condition was therefore clinically one of chronic hypertrophic laryngitis. February 20th the tube was coughed out for the first time in her history and also for the first time in two years she breathed three days unassisted. Then the tube had to be reinserted on account of increasing dyspnœa. This comparatively slow recurrence of the stenosis often occurs during recovery. It is too rapid to prove an error in diagnosis or that the condition is cicatricial. February 28th a larger special rubber tube was inserted, and special tubes of increasing size were used with infrequent changing until July 8th. At this change, when the largest of the special tubes had been worn a month, the condition seemed as bad as ever, and acting under the belief that a spasm of the adductors of the larynx might have something to do with the difficulty a normal four-to-five tube was inserted until July 16th. On removing the last or normal tube I found the breathing, though somewhat difficult, was fairly good and hence the supposition that the obstruction at the last change was due to a cramp of the adductors and atrophy of the abductors seemed correct. I therefore returned to the special metal tube, which had produced the first improvement in March. July 27th this tube was removed and cleaned by the house surgeon, as it seemed to be foul and producing a cough. Respiration, though difficult, was possible. In August a nephritis developed and this was followed by a chronic pneumonia, which at autopsy was pronounced by the pathologist to be simple and not tuberculous. August 26th the tube was removed and natural respiration for the first time was easily possible, but the dyspnœa gradually returned and on August 29th the tube had to be replaced. The child continued to gradually lose ground and finally died from the pneumonia on September 12th. The tube was not removed until after death, but then the larynx appeared

[1] The twenty-two histories which it was the author's intention to submit in detail made this article so long that at the request of the editor only those are presented which give some idea of the worst difficulties and the method of treatment. In consequence these convey a more gloomy impression of the prognosis than is warranted by the majority of the histories.

open enough for respiration to have been carried on unassisted. The report on the specimen will be found in the first part of this paper.

CASE VII.—*Stenosis due to hypertrophic laryngitis following intubation for laryngeal diphtheria; complicated by cicatrices resulting from several laryngotomies and tracheotomies; obstruction from spasm of the adductors; also from granulations; cured by two and a half years of treatment with "special" tubage; duration of stenosis, six years.*

M. M. was admitted to the Willard Parker Hospital June 2, 1896, at the age of three years. She had been intubated six days previously for diphtheria. The stenosis persisted and on account of frequent and dangerous autoextubation she was tracheotomized July 13th. The stenosis still persisted and intubation was again tried. Astringents like alum applied on the outer surface of the tube to the laryngeal mucous membrane, as advised by Dr. O'Dwyer and afterward curetting the larynx through the mouth, were tried without success. In October, 1896, transferred to the Metropolitan Throat Hospital and thyrotomy performed by Dr. Wagner. Some thickened tissue removed from the interior of the larynx and the wound packed. Breathing through the tracheal cannula. The only pathological change noted during the operation appeared to be a general thickening and hypertrophy of all the intralaryngeal soft parts. In October, 1898, another thyrotomy, performed by Dr. Hope, with division and excision of numerous cicatrices. In January, 1900, the patient, then seven years old, was transferred to Gouverneur Hospital still breathing through a tracheal cannula. Under chloroform a much deformed larynx, which barely admitted one of the smallest urethral sounds, was dilated from below through the tracheal fistula with urethral sounds up to No. 25 F. There was a "spur" opposite the fistula and the entrance to the trachea below was contracted around the cannula; this was also dilated like the larynx. A ten-to-twelve normal intubation tube was then inserted and subsequently changed every four or five weeks for cleansing. In May, 1900, the tracheal fistula finally closed and so cut off all respiration except through the larynx and the stenosis was still marked. At this change I made several attempts at intubation and failed on account of the deformity and displacement of the larynx due to the numerous cicatrices. The consequent traumatism produced complete obstruction and asphyxia was barely avoided by a hasty tracheotomy, after which intubation was finally accomplished. In July, 1900, as a preliminary to changing the tube and to avoid the previous danger, I again punctured the trachea and then intubated without difficulty. This largest-sized tube was changed at intervals of four weeks without perceptible gain and therefore with the object of obtaining the utmost dilatation a "special" tube was employed of the same length as the normal but of

much larger diameter. This was worn with the usual changes until January 15, 1901, when it was removed and a cure seemed at last obtained, but five days later at night there was a sudden attack of dyspnœa and the child was barely saved from asphyxia by another hasty tracheotomy by the house surgeon. Some infection followed with recontraction of the cicatrices, and not until March was intubation again practised. A little later dyspnœa began to reappear with the tube in place. An exploration revealed exuberant granulations at the base of the epiglottis overtopping the tube and blocking its lumen. In July another thyrotomy had to be performed to gain access to the obstruction and the granulations were then scraped and cut away. A normal tube with a small head to avoid pressure on the ulcerated surfaces was inserted at the close of the operation. This was worn until no more bleeding occurred at the periods for cleansing. In November the special tube with the large diameter was again used and worn with occasional removals for cleansing until February, 1902, when a cure seemed obtained, but that night there was a sudden attack of dyspnœa and another tracheotomy by the house surgeon. On the following day the large calibred special tube was reinserted and worn until May. Then a normal tube of the eight-to-nine size was substituted for two weeks, and in June the patient was at last dismissed cured. She has remained well and free from dyspnœa ever since and even has a good though husky voice. She is at present attending school (October, 1905).

CASE IX.—*Stenosis due to hypertrophic laryngitis following intubation for laryngeal diphtheria; thyrotomy followed by sloughing of the cartilages; permanent cure after three months of proper treatment.*

I. B., aged four years, admitted with diphtheria to Willard Parker Hospital and intubated in December, 1896. The stenosis persisted and necessitated retention of the tube. Frequent and dangerous autoextubation began, and to prevent asphyxia tracheotomy had to be performed on February 20th. A month later intubation with a normal four-to-five tube was practised with the tracheal cannula in place. No autoextubation occurred, so the cannula was removed and the fistula allowed to heal. The complete stenosis persisted and she was transferred to Gouverneur Hospital October 15, 1897. A week later the tube was coughed out and a hasty tracheotomy performed by the house surgeon. In November the cannula was removed and intubation again continued. January 10, 1898, as respiration was impossible without the tube, I explored the larynx by a median vertical incision through the thyroid and cricoid cartilages. The tissues were thickened and soft throughout the whole interior of the larynx, in which there were no ulcerations and no cicatrices. By bringing the parts together it was found that the hypertrophied mucous membrane completely occluded the lumen of the larynx. With the idea of producing a vertical cicatrix on

each side of the larynx and so retracting the swollen parts I passed a silk suture through the cartilage and thickened mucous membrane on each side and tied it on the outer surface of the corresponding thyroid ala. This retracted the tissues sufficiently to allow easy breathing and the larynx was closed, but infection followed and in the course of the next few weeks apparently the greater part of the thyroid and cricoid cartilages sloughed out. April 20th the patient was discharged seemingly in a hopeless condition and condemned to the permanent use of a tracheal cannula. In August the patient returned with a dense cicatrice occupying the position of the larynx, but under chloroform with some difficulty a probe was passed from the tracheal fistula up into the mouth. This was followed with sounds of increasing calibre until the larynx and the entrance to the trachea had been dilated up to No. 24 F. A normal four-to-five tube was then inserted, the cannula removed and the fistula allowed to close. On October 1st the tube was removed and a six-to-seven normal tube substituted. The stenosis was still nearly complete. At the next change, in November, natural breathing was found possible and the tube was left out. For the first two weeks there were several attacks of inspiratory dyspnœa, especially at night, but under the administration of a little morphine these gradually disappeared. Since then the cure has remained permanent and the voice is fairly good.

CASE XV.—*Stenosis from cicatrices of syphilitic origin; clogging of a metal intubation tube; extreme subsequent contraction of the larynx which required laryngotomy to insert a plugged tube; apparent cure followed by apparent failure owing to unrecognized spasm of the adductors.*

C. McD., aged twenty-five years, was admitted to Gouverneur Hospital August 18, 1896, suffering from marked laryngeal dyspnœa, which had been increasing gradually since the previous April. There was an indistinct history of syphilis. She was immediately tracheotomized and, as is usual in emergency cases, the cricoid cartilage was divided. Subsequent examination revealed a larynx much deformed and obstructed by cicatrices. There was no ulceration. On November 15, 1898, under chloroform the larynx was dilated up to No. 35 F., with urethral sounds entered through the trachea and a metal tube inserted. Much difficulty in swallowing followed and a week later I arrived just in time to extract the tube, which had become nearly occluded by dried mucus. Another intubation was refused and the tracheal cannula had to be inserted. Attempts to use a Dupuis' segmented right-angled cannula failed, owing to the difficulty in swallowing and to the pain and swelling in the top of the larynx over the upper end of the superior half of the instrument. There was the same difficulty with an ordinary curved cannula, to which was fitted on its upper surface a bivalve dilating attachment to extend up into the larynx. Intermittent dilatation

with Schroetter's tubes proved useless, as is usual in these bad cases. The patient left wearing a tracheal cannula. In February, 1899, she returned and under chloroform the larynx was found almost but not quite occluded. The thyroid and remains of the cricoid cartilage were then split in the median line upward from the cicatrix and the tortuous line of mucous membrane representing the remains of the lumen of the larynx was laboriously followed with a fine probe, as in external urethrotomy without a guide, until the pharynx was opened. A small adult intubation tube was then inserted through the mouth, and after making longitudinal incisions in the most prominent cicatrices the wound was sutured over the tube and a tracheal cannula placed in the lower angle beside the tube. A couple of weeks later an intubation tube with its anterior half cut away to receive the inner end of the cannula was attempted, but clogged with dried mucus and pus so often that it was abandoned and a "plugged" tube was substituted. This worked fairly well, but the fistula in healing came to be located about the middle of the thyroid cartilage. It, however, brought the tube near the surface and the patient could clean it daily with a feather after removing the plug. A change of the tube every four or five weeks demonstrated rapid improvement, and in May the tube was removed; the fistula in the neck contracted almost immediately, so that at the end of twenty-four hours it was practically closed. Meanwhile natural respiration was carried on without the slightest difficulty, but suddenly three or four days later, at night, an attack of dyspnœa almost caused asphyxia (adductor spasm), but fortunately enough of the fistula remained to enable the house surgeon to dilate it and insert the cannula. Further treatment was then refused for some time and the patient went home wearing the tracheal cannula. April, 1900, under chloroform, the contracted larynx and trachea were dilated as before. But so much difficulty in intubating was experienced on account of the "spur" which had formed in the posterior wall of the trachea opposite the fistula, that the attempt had to be postponed until a small right-angled blunt retractor, grooved on its convexity to fit the intubation tube, could be constructed. A week later, under chloroform, with this instrument in the lower angle of the fistula to drag the trachea forward and to guide the tube in the convexity of the retractor into it, intubation was accomplished. The tube used was one with a "plug" attachment. This was kept in place with occasional changes for cleansing for about four months or until August, 1900, then a cure seemed obtained, but two days later the fistula, having nearly closed, there was a sudden attack of dyspnœa as before at night and the cannula was reinserted and the patient refused further treatment. When seen last about a year later she was pregnant and showed signs of advanced pulmonary phthisis and has probably died since then, but at that time, though the cannula was still worn, laryngeal res-

piration, much to my surprise, was fairly good and another course of intubation would probably have produced a cure. But it seemed hardly justifiable in her condition. The apparent lack of success after prolonged dilatation was undoubtedly due to my failure to recognize and allow for the "cramp" of the laryngeal adductors. A tube with a small neck worn for a couple of weeks after the removal of the large ones would have permitted the abductors to regain their strength and so prevented the cramp or spasm of the adductors. She was a very nervous woman, somewhat addicted to beer and easily terrified, so that the sudden dyspnœa after apparent success would make her refuse trust in anything but the cannula. In the light of later experience I have regarded this case as really a success, though apparently a failure.

All the credit for the main principle in the management of chronic cicatricial laryngeal stenosis, that of long-continued dilatation with an intubation tube, of course belongs entirely to the late Dr. O'Dwyer. He long since insisted that this method was the only one generally applicable, and he published several cases of chronic stenosis due to one or another cause which were brought to a satisfactory conclusion by intubation continued for several months. But he had no regular course of procedure for curing the occasional cases of "retained tube" with chronic hypertrophic laryngitis, which in addition are often complicated by dangerous autoextubation, nor did he go into the details of the management of the various conditions accompanying stenosis with a retained tracheal cannula, and in these small particulars the histories of the cases given above supply much that is valuable. For there is no more serious disability than chronic respiratory obstruction in the upper air passages, and any method of treatment which is safe and offers a reasonable prospect of cure in nearly every instance should be welcome. Great patience, perseverance, and foresight with some technical skill are required, but given these my experience has led me to believe that a failure to ultimately restore natural respiration and even voice must be exceptional. Intermittent dilatation with Schroetter's instruments and all the various operative procedures which from time to time have been recommended chiefly in German literature are notoriously unsatisfactory. Some conditions must, however, occasionally be encountered which can only be remedied by plastic or other more or less extensive surgical interference, but they are rare and even then subsequent cicatricial contraction will probably arise. Primary union in the several laryngotomies mentioned above was much less common than suppuration, which may vary from simple failure of the wound to unite by first intention to extensive cellulitis of the neck with lymphadenitis and sloughing of the cartilages. In short, operations for stenosis of the larynx or upper trachea are seldom advisable except for the removal of tumors. Papillomata are probably the most common of these, and several

cases have been reported in which intubation alone was said to have caused their disappearance. But without the definite knowledge of the extent of the disease only to be gained by a previous thyrotomy or tracheotomy intubation may prove dangerous; the papilloma or any other growth may be caught in the point of the tube and masses of this tissue pushed down ahead of it into the trachea like a gun wad. Several years ago I encountered a death from asphyxia in the practice of a colleague produced in this way. Hence, of course, intubation, like any other surgical procedure, cannot be indiscriminately employed, and an open operation, if only to make sure of the conditions, may sometimes be a necessary preliminary. But aside from the inspection or removal of new-growths the causes of stenosis rarely require anything beyond dilatation followed by intubation.

These cases can therefore generally be grouped as obstruction due to (*a*) cicatrices, (*b*) hypertrophic laryngitis, (*c*) granulations or œdematous ulcer, and (*d*) spasm of the adductors. One or more of these conditions may coexist and before proceeding to the treatment it is necessary to differentiate briefly the symptomatology. A cicatrix is easily recognizable by the history of a traumatism or disease with or without ulceration, coupled with inspection and instrumentation of the larynx, or tracheal fistula if one has been made. The deformities common after tracheotomy have already been described. The chief difficulty lies in differentiating between some of the tumors, including the granulomata and the persistent stenosis—hypertrophic laryngitis—sometimes following intubation for an acute condition, generally laryngeal diphtheria. The tumors, of course, if they cannot be removed through the mouth, require open operation any way, and exploration is generally safer than working in the dark. But the differentiation of a cicatrix from the other common conditions should be simple even when an open ulcer overlies it. In the first place it offers considerable resistance to instrumentation, and after dilatation or intubation the stenosis returns gradually within days or weeks. An hypertrophic laryngitis offers no resistance to the insertion of a tube, and after its withdrawal the recurrence of the obstruction is immediate and often complete. When these two difficulties occur together, as is not infrequent, especially after diphtheria, the history coupled with the resistance to instrumentation and the sudden recurrence of dyspnœa after it, should present no great difficulties, although any traumatism of the larynx may give rise to an œdema of quite sudden onset. The spasm of the adductors, though not strictly speaking a cause of chronic stenosis, so closely simulates clinically an hypertrophic laryngitis that it must be included in the differentiation. It seems to have been recognized long before the days of intubation and so must occur whenever the laryngeal muscles have been for any length of time at rest. Of course, when an intubation

tube has been worn for a considerable period, particularly the "special" instrument which I recommend with an abnormally large neck, the abductors are put out of employment and must undergo an atrophy of disuse while the adductors are under some more or less constant tension with every attempt at coughing. However the explanation may be made, clinically at least there is no difference appreciable between spasm of the adductors and hypertrophic laryngitis after removing the intubation tube or plugging the tracheal fistula. When, however, this condition is suspected in a patient who has been intubated the diagnosis can sometimes at least be made by extubating under an anæsthetic. This removes the element of fright and abolishes the disturbances somewhat vaguely termed "reflexes," and normal respiration may be discovered where it might not otherwise be anticipated. Furthermore, a sudden spasm, and that, too, of very dangerous dyspnœa, generally occurring at night with coughing, and inspiratory rather than expiratory, can pretty safely be ascribed to this cause. The resistance encountered in this condition by the tip of an intubation tube catching on the closely approximated vocal cords should not be misinterpreted as a cicatrix, which takes time to develop and contract. The treatment of the two conditions is very different. "Granulations," if not perceptible to sight or touch, bleed easily upon instrumentation and generally occur as the result of the pressure or irritation of an intubation tube or cannula, in which they produce a more or less gradually increasing obstruction either to inspiration or expiration, seldom to both acts in succession. A spasmodic cough accompanied by more or less hemorrhage and dyspnœa may also give some indication of their presence; but I am convinced that they are a far less frequent cause of chronic obstruction than seems generally to be believed. When there is bleeding accompanied by stenosis following extubation or decannulization it is much more reasonable to ascribe the dyspnœa to a simple œdematous ulcer overlying an hypertrophic laryngitis or a cicatrix and not to exuberant granulations. The slight traumatism incident to the relief of the pressure of an intubation tube against such an ulcer or the "gagging" which often accompanies the removal of a cannula, especially if it is high up, can produce enough congestion in an eroded area to cause it almost immediately to nearly or quite occlude an already narrow space. I have never been able to positively verify such a condition but believe in its occurrence, and though it may be impossible to differentiate from a pure hypertrophic laryngitis, which is also very vascular and bleeds at the slightest touch, the treatment does not differ.

In a former article on this subject I recommended the long-continued use of as large a normal intubation tube as the larynx would admit, but experience has caused me to modify this treatment very materially for the different conditions. Simple chronic hypertro-

phic subglottic laryngitis is probably the most common cause of obstruction after intubation. This ought to read "after diphtheria," because it seems unfair to imply that it is due to intubation inasmuch as it was recognized as one of the most frequent obstacles to the removal of a tracheal cannula long before intubation was invented.[1] It may also occur entirely independently of any acute disease, and Case I. seems to be an example of this. There will, however, be considerable difficulty in convincing most intubators that unskilled or rough instrumentation does not have something to do with its inception or continuance, for a certain number of these cases will get well in the course of days or weeks with careful and frequent change of a tube, smaller if need be than the development of the patient would ordinarily indicate, the object being to minimize traumatism. Local astringents, as advised by Dr. O'Dwyer, applied to the interior of the larynx in a coating of gelatin or other substance adhering to the outer surface of the tube, may also succeed. But there are a number of cases (about 1 in every 100 intubated as estimated from the statistics given in my previous paper[2]) which will resist all these efforts for their relief. If the stenosis of this character has persisted for about a month in spite of all the usual plans of treatment, and spasm of the adductors can be excluded, another method must be adopted. Some of the patients will recover if as large a normal tube as can pass the cricoid cartilage is used for five to six months and changed at intervals only for cleansing. At the end of every four or five weeks the tube becomes foul and causes a bad odor in the breath and considerable cough. But usually in a persistent case it is better to start with a "special" tube or one having the neck one or more thirty-seconds of an inch larger in the transverse diameter than the normal instrument, with the object of applying pressure upon the thickened area within the larynx. As this area generally extends from above the vocal cords to or through the cricoid cartilage, and as the retaining swell of the tube must be enlarged with the "neck" to prevent autoextubation the retaining swell of the special tube must be enlarged along with the neck; and the same must be done with the head or there is danger of the instrument slipping below the epiglottis, which will then more or less completely occlude the upper aperture. Practically in these cases I am accustomed to make trials with normal tubes of increasing calibre under complete anæsthesia if necessary, until I have found the largest tube which will enter and pass through the larynx with some slight force. The "special" tube is then made of the length of the normal tube proper for the age and development of the patient, but with a retaining swell having the diameter of that of the trial tube. The neck must then be at least three-thirty-seconds of an inch smaller than the retaining swell and the head six-thirty-

[1] Köhl. Archiv. of klin. Chir., 1887, Band xxxv. p. 75.
[2] Annals of Surgery, May, 1905.

seconds of an inch larger than the retaining swell. If a metal tube is to be used its weight is better and more safely sustained by a head which is at least seven-thirty-seconds of an inch larger than the retaining swell and a large tube should have eight-thirty-seconds of an inch difference. If the child is under four or five years of age and so needs the smaller sized tubes the thirty-seconds of an inch difference should be altered to sixty-fourths of an inch. Moreover, a difference of three-thirty-seconds of an inch between the neck and retaining swell is the minimum difference which I have found by experience sufficient to keep the tube in place. Some patients will require four-thirty-seconds or even five-thirty-seconds of an inch difference. In the first part of the treatment of an hypertrophic laryngitis, especially if the stenosis is complete, as is frequently the case, it is wise to start with a special tube having a diameter of the neck five-thirty-seconds of an inch less than that of the retaining swell. Care in these seemingly minute details may mean all the difference between life and death, as unexpected autoextubation or aspiration of the tube into the trachea (impossible with the normal tube) can easily be fatal accidents. After a special tube thus carefully made has been worn a month or six weeks it usually must be changed and cleaned or the accumulation on it may cause it to clog suddenly, or produce ulceration, or a septic bronchitis, or pneumonia. Another tube should be at hand of one-sixty-fourth or one-thirty-second of an inch larger diameter through the neck and retaining swell than the last. Some slight improvement in the stenosis can generally thus be detected in the resistance offered to intubation if it is not clinically apparent. The judgment of the operator must determine the amount of presure which can be borne without sloughing. Fortunately the larynx seems capable of bearing much more traumatism in these chronic conditions than one would think possible, and if some little ulceration occurs as it often must, the epithelium will in course of time cover the raw area unless the pressure is extreme. Furthermore this ulcerative process, if not too extensive, may actually be the way in which cure takes place in these cases of simple hypertrophic laryngitis. Scattered areas of fibrous cicatricial tissue may in contracting against the cartilages do away with the preceding chronic exudative and productive inflammation. A most important question in these and to a less extent in cases having another etiology lies in the use of metal or vulcanized rubber tubes. The metal instrument is usually made of bronze or some less expensive compound heavily gold plated. At any rate it is composed of two different metals, the exposed part being gold. In two or three of the cases of hypertrophic laryngitis described above absolutely no progress could be made after weeks or months of treatment with hard rubber tubes so constructed as to make all the pressure the larynx seemed able to bear. Then after the substitution for the special rubber tube of a similar or

slightly smaller compound metal tube, either a cure or very marked improvement was found at the next change or within four or five weeks. I am unable to explain this fact except on the theory of some electrolytic action. The metal instrument is far heavier than the rubber and the pressure may have something to do with its advantages, but I doubt it, as I know I have made more pressure with a large rubber than with any metal tube and there must be some other reason for the difference. The claims for electrolysis are generally regarded with suspicion, but these metal tubes, after being worn a month or two, become heavily coated wherever they press most upon the larynx, that is chiefly around the head and neck, with a stony, calcareous deposit, and under and around this the gold plating entirely disappears. There is of course no acid or alkali in the body which can dissolve gold, and it does not chip off but apparently dissolves. The external calcareous deposit of course roughens the surface, and if the tube is worn for a long time may reach such proportions as to prove a very serious obstacle in extraction. I once met with such a case in the practice of a colleague in which the tube, having been in place for several months, required for removal general anæsthesia and a strong pair of forceps to permit a firm grasp of the head of the tube. The metal tube will, however, if so constructed as to exert moderate pressure upon all the thickened tissue of an hypertrophic laryngitis, sooner or later produce a cure. But the patient who wears one must be constantly watched, as it has a tendency to clog and thus asphyxiated one of my cases and very nearly did the same with another (Case XV.), and naturally after those experiences I have always been afraid of these tubes and only resort to them when the special hard-rubber instrument after thorough trial in increasing sizes proves a failure.

Autoextubation is a frequent and very dangerous complication in any intubated case, but especially dangerous in hypertrophic laryngitis and introduces the "special," or normal "clamped," or "plugged" tubes. Some patients will cough out any tube which has not such a mechanical attachment to keep it in place, and as autoextubation is most common with hypertrophic laryngitis, I have come to believe that some degree of this condition is, if not the primary, at least the secondary cause of the difficulty. The chronically inflamed tissues are irritable and their contact with the tube excites coughing accompanied by retching or involuntary expulsive contraction of the pharynx. The combination of an attempt to cough and vomit at the same time seems the surest way for the patient to get rid of the tube, and life generally depends on its immediate reinsertion or a hasty tracheotomy. The latter operation probably, by giving rest to the larynx, seems occasionally to produce a cure, but ordinarily it only adds a cicatrix and a possible tracheal deformity to an already bad-enough condition. If there were no other way to escape the dangers of autoextubation it might

be justifiable, but hypertrophic laryngitis used to be one of the most frequent obstacles to decannulization and it seems hardly proper to inflict a tracheal fistula for its cure. Still, the tube must be kept in the larynx and for this purpose I offer the "clamped" or "plugged" instrument. If autoextubation is at all frequent the trachea is carefully opened in the median line through the second and third rings and the intubation tube while in position marked to correspond with the incision, or if a tracheal fistula has already been made the tube is inserted and marked at a point coinciding with the fistula. During the few succeeding days required to make the instrument the cannula will have to be worn, but this inflicts the least possible damage. Some explanation is required as to the comparative advantages of a "clamped" or "plugged" tube and as to the use of such an instrument in general. It is assumed that the trachea is opened carefully in the median line well below the cricoid, and only enough to comfortably admit the cannula and subsequently the retaining attachment for the tube. Deviations from these rules as indicated previously promote cicatrices and tracheal deformities. If the fistula is already existent when the patient comes for treatment, as is frequently the case, there is of course nothing to be gained and much may be lost by enlarging or altering the position of the fistula, and it is then only a question of a normal or "clamped" intubation tube. The self-retaining instrument is far preferable to a tracheal cannula, to which it is the only alternative in hypertrophic laryngitis complicated by frequent and dangerous autoextubation if only because it places the patient in a practically normal condition with the single exception of a whispering voice. For less serious cases of stenosis, especially those which experience has demonstrated can breathe for an hour or two without mechanical assistance, the self-retaining tube may be thought to possess doubtful advantages. But in these there are many factors to be considered. To be sure at least one of the patients in this series could not swallow while wearing the instrument, but that experience is very exceptional, and in adults, especially "nervous" subjects, equally common with the normal tube. That the clamp or plug may act like the cannula in promoting or maintaining a tracheal deformity or favor a necrosis of cartilage has always been my fear, and hence I never use it except in cases of necessity; but experience seems to show that no ill effects are to be anticipated, and hence I have no hesitation in recommending it whenever there is the least anxiety as to autoextubation or subsequent adductor spasm. In my earlier cases, owing to unwarranted suspicion of the self-retaining tube, I believe I have taken great and improper risks in using the "special" tubes, although they are less unsightly and more ideal in effect. The perpetuation of the fistula by the "plug" also offers the distinct advantage of a sense of security in adults who are relieved of the dread of autoextubation, and in case of

clogging of the tube can remove the plug and cleanse the interior of the instrument by simply poking a feather through the opening. Indeed I was first forced to employ this kind of tube in Case XV.; the normal (metal) tube previously used became obstructed one day quite unexpectedly and so nearly produced asphyxia that a substitute arrangement seemed imperative. This woman thereafter cleansed her tube in the way suggested every morning and never again had difficulty.

After removal of any tube, particularly a large or special one which has been worn a long time, there is great danger of sudden adductor spasm and strangulation and an opening in the trachea already present is a great safeguard. The spasm, however, usually occurs some days or hours after the removal of the tube (and plug), and the "fistula," when not kept patent artificially, is apt to nearly or quite close before this dangerous incident. The usual curved cannula, which could be employed in the interim, is manifestly improper, as it drags the lower segment of trachea forward and pushes the upper back, and therefore I recommend a perfectly straight cannula so fitted to its shield that it can be pushed in or pulled out and thus stay with its inner extremity protruding into the lumen of the trachea. The diameter of the straight cannula should not be over one-eighth or one-quarter of an inch to avoid undue separation of the tracheal rings and approximation of their unsupported posterior extremities, and at the same time it must be large enough to permit, if need be, the insertion of a tracheal dilator through the fistula it maintains. This straight cannula only need be worn for the first two weeks after removal of the intubation tube. Subsequently to this period I have never met with an adductor spasm, and any other obstruction occurs slowly and can be met by reintubation.

The plugged tube cannot be made in small sizes, as either the plug or the tube or both must be constructed of vulcanized rubber, and there is a limit to the strength of this material. The plug and tube cannot both be made of metal, as the calcareous deposit which always takes place on the surface of a metal tube is very hard and firmly attached and might easily prevent the removal of the plug if the latter were likewise made of metal. The workmanship must also be of the best and very exact, because the least irregularity in correspondence between the lumen of the plug when screwed in place and that of the tube will cause rapid clogging. Possibly a metal tube with a hard-rubber plug is the best combination for cheapness, strength, and durability, and as the retaining rod and cap are also of metal it offers possibilities for attempts at electrolysis for those who believe in such treatment. The "clamped" tube is the only one I have found capable of construction in the small sizes for children under ten years, and it is very useful. The tube has to be grooved on its outer surface to smoothly and exactly

receive the clamp, as any irregularities or roughness will produce ulceration. The tube must be of rubber and the clamp preferably of silver, as a metal tube and a metal clamp would speedily become inseparable on account of the calcareous deposit previously mentioned. Also a clamp fashioned like an obstetric forceps is much better than one having the blades pinned together as in a pair of ice tongs. The disarticulated blades, though somewhat difficult to insert in a squirming, frightened child, will enter a very small hole and also there is no pin to break and drop into the trachea. The "cap" of metal then screws down snugly on the tube over the shanks of the clamp and should leave no rough or irregular interval to produce irritation.

The treatment of the adductor spasm, which is particularly to be dreaded after using large or "special" tubes for the cure of an hypertrophic laryngitis, has been partly outlined in the advice given as to maintaining the patency of a tracheal fistula. It is dangerous and must always be watched for after any prolonged intubation; as it is apparently due to an atrophy of disuse in the abductors a small tube which permits them to functionate and at the same time maintain respiration is a logical procedure. After determining, if necessary, with the aid of anæsthesia that the hypertrophic condition has subsided a normal tube of the size proper for the age and development of the child is inserted and left for about one or two weeks. During this period and for a week longer assistance must be constantly near at hand in case of autoextubation. If, however, a clamped tube has been used and the small straight cannula retained in the fistula the danger is very slight. The nurse or any attendant, should difficulty occur, can readily push the straight cannula or a curved one for that matter into the trachea. The straight cannula is apt to slip outside the cartilaginous rings, especially when worn with an intubation tube. A curved cannula, by the way, inserted alongside the tube or into a fenestrum in its anterior wall, I have found to be an utter failure.

A *cicatricial* stenosis, particularly if a hasty tracheotomy has at some earlier stage been performed, is often complicated by a contracted trachea with a spur opposite the fistula; and if there has been a tracheotomy for the autoextubation following laryngeal diphtheria, there is in addition probably more or less hypertrophic laryngitis. But even when there is a history or probability of considerable loss of cartilage (see Cases VII., IX., and XV.) and contraction of the larynx until the lumen seems practically obliterated (Case XV.), experience has shown me that the prognosis is still good. In the milder cases without a tracheal fistula insertion of a normal hard-rubber intubation tube for from one to six months will invariably effect a cure. Cocaine is a sufficient anæsthetic, and if the resistance to a tube of the proper size seems too great a smaller one can be employed for a few days and then a larger sub-

stituted. In the tighter and more deforming strictures general anæsthesia is required to permit the necessary preliminary dilatation. The bad strictures always have had a tracheotomy at some earlier period or need it urgently when first seen, and the fistula serves admirably for the introduction of urethral sounds which are excellently adapted for the purpose. Beginning then with the smallest size, which will pass through the contracted area, others are passed in rapid succession until the intubation tube can be inserted. Some judgment must of course be used to avoid the possibly fatal accident of making a laceration which may extend into the pharynx or œsophagus, but though I have done the operation many times and used very considerable force I have never encountered this or any ill results. Nevertheless the possibility of its happening must not be overlooked. Occasionally, as in Case XV., the stricture may be so tight or tortuous that its lumen may have to be opened by a careful dissection like a strictured urethra, but as long as any traces of mucous membrane are left and a tube can be passed from the pharynx into the trachea over or through the remains of the epithelium, there can be good hopes of a cure. Even very extensive loss of cartilage, as in Case IX., may be repaired, although Dr. O'Dwyer[1] cites one instance in which as the result of diphtheria the whole cricoid had sloughed out and this he considered irremediable except by tracheotomy. I should not, however, be inclined to condemn such a case without a trial.

In addition to the stricture, which is generally worse above the fistula, there will always be one at and just below this point, especially if the cannula has been worn long, and this also must be dilated like the one above. Then when inserting the tube the spur opposite such an old fistula in the posterior tracheal wall will be a serious obstacle to pass unless a small right-angled blunt retractor or hook, grooved on its convexity to receive and guide the entering tube downward and backward, is placed in the lower angle of the fistula, dragging the inferior tracheal segment forward. In adults I have several times had to exert all the force possible in two fingers pushing down from above on the head of the tube while counter-pressure was made externally on the larynx with the other hand to pass this point with its spur.

The comparative advantages of using a normal, a special, or a "clamped" or "plugged" tube in these cases have already been discussed. As a rule a combination of a plugged with the principle of a special tube is the best. The plug is a safeguard against clogging and autoextubation and maintains the fistula as a subsequent escape from the difficulty of adductor spasm; the feature which for brevity I have called "special" in the instrument means an enlargement of some portion of the diameter over the corresponding diame-

[1] American Surgery, May, 1900.

ter of the normal tube to exert a dilating pressure upon some particular part of the larynx or trachea. The normal tube is long, as it was designed by Dr. O'Dwyer to pass as deeply as possible into the trachea beyond any diphtheritic process. Its lower end is constricted from side to side to pass harmlessly through the glottis, the retaining swell is as large as will pass easily through the ring of the average cricoid, and from this point upward the tube is again flattened from side to side to fit the larynx. The "special" tube must always have the retaining swell, and this can be larger than the normal, as a diphtheritic swelling within the cricoid and consequent sloughing from traumatism at this point need not be considered; but at its neck or its lower end its diameter must be increased to stretch the stricture to be combated up to the largest possible normal lumen of the air passages. No exact measurement for this can be given, and they can only be approximately estimated and by a trial of different tubes. If, for example, the stricture is in the trachea at the lower end of the cannula (rare, though I have seen one case at the Willard Parker Hospital), the tube from the retaining swell down must be of the same diameter throughout and long enough to pass the contracted area. Again, if there is an hypertrophic laryngitis complicating a cicatrix the tube must have the diameter of its neck increased, although (unless it is a clamped or plugged tube) the transverse diameter of the retaining swell must always exceed that of the neck by three-thirty-seconds or preferably four-thirty-seconds of an inch.

A rough estimation of normal measurements which I have found useful is obtained by comparison with the patient's index finger, which is somewhere near the calibre of his trachea below the cricoid. The length of this finger is also about the distance from the suprasternal notch to the tracheal bifurcation.

The prognosis of a cicatricial stricture is difficult to give. Some cases seem to recover permanently after a few weeks or months of continued dilatation; others, especially those in which the rules for a proper tracheotomy have not been or could not be carried out, will relapse from time to time and prove very discouraging.

In a few instances I have attempted to dissect out all cicatricial tissue and, by accurate suture of the trachea and overlying soft parts over a well-fitting tube, to obtain primary union and so do away with these annoying and dangerous relapses. But the results have not been encouraging, possibly because asepsis is practically impossible. In Case XI., although the wound united throughout by first intention, the stricture was again evident within a few days after the removal of the tube. In the others infection took place and the wound broke down in whole or in part. On the other hand, persistence in the dilatation for months or years (Case VII.) has seemed finally to yield a permanent cure, and hence in general cicatricial strictures of the larynx or trachea should be continuously

maintained for at least six months. An attempt may then be made to do without the tube (with proper regard to adductor spasm), and at the end of a week the larynx and trachea tested for recontraction by insertion of the tube last worn. If none is apparent the test should be repeated once every one or two weeks for a month or more. At the first sign of recontraction the tube should be inserted and worn for another six months and then again tested.

But if the stricture is once allowed to contract seriously beware of attempting to intubate without preparations for tracheotomy. Such a recurrent stricture, especially if near a previous tracheal fistula, is apt to be very tight before it causes distress and very resistant to tubage. It is wisest to first attempt a passage with the Schroetter dilators, and if these fail do another careful tracheotomy in the old cicatrix when present and then dilate again with urethral sounds followed by intubation, as at first. Perhaps the safest rule after a cure seems obtained in these cases is intubation for a week every couple of months or oftener if required for several years or until no tendency to contraction can be detected during a considerable period. A pure hypertrophic laryngitis is, if anything, more difficult to handle than the cicatricial form of obstruction, as there is no time to think in the interval of changing the tube—generally for weeks or months the obstruction remains complete and the clean tube has to be immediately reinserted and it takes longer to cure the condition. But after it is once well I have never met with any recurrences of the stenosis. The average duration of treatment has been from about six months to a year, although one case recovered permanently within four weeks and another, complicated by numerous cicatrices to be sure, required nearly three years before natural breathing was finally obtained. This very long period was, however, largely due to my inexperience at that time.

Granulations as a cause of chronic obstruction are, I am convinced, rare, and when present are fortunately easily handled. Their removal, like any other new-growth, by cauterization, excision, or curettage is of course the first step and this can generally be accomplished through the mouth or tracheal fistula, although the latter may have to be enlarged to provide access. To prevent recurrence sources of irritation must be eradicated by making a new fistula or by changing the shape or length of the tube.

Granulations are most common at the base of the epiglottis around the head of the intubation tube, and Dr. O'Dwyer describes two cases in which they were finally overcome in this situation by so building up and remodelling the head of the instrument as to distribute the pressure it exerted over a larger area. In Case IX. the difficulty was satisfactorily met by using a smaller than normal tube. The principle of course is to remove the traumatism, or at least minimize it, and thus the details for individual instances can be readily adjusted.

A word as to feeding may be necessary. For the first few days after intubation liquids only are permissible and they should be taken in the Casselberry position with the mouth lower than the pharynx and it lower than the œsophagus. But it is surprising how quickly these patients learn to swallow a varied diet naturally and how few abnormalities they display. Even if an intubation tube should have to be worn permanently this is far preferable to the alternative of a tracheal cannula.

EMPYEMA OF THE FRONTAL SINUS.[1]

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The case I report illustrates so well the so-called closed empyema of the frontal sinus and certain peculiar phases of the disease that I have thought it might prove of interest. By closed empyema I mean that at no time could a discharge of pus into the nose through the frontonasal canal be detected. Though several careful examinations were made from time to time, this most important symptom of disease of the accessory cavities of the nose did not appear. The history of the case is as follows: The patient is now thirty years of age. He had most of the diseases incident to childhood, and since then has been healthy until the beginning of his present illness. His parents are living at advanced ages, and are healthy. In January, 1904, he had what he considered a mild case of "grip." He felt weak and languid and was forced to lounge about his room for two or three days. After this indisposition he convalesced rapidly, and was soon able to resume business. About two weeks later he was taken with a severe pain over the left eye. The pain recurred regularly every other day, and was peculiar in that it would begin at twelve o'clock noon, or between the hours of 12 and 4 P.M. If it did not appear before five o'clock in the afternoon the patient had nothing to fear until the next day. Occasionally the pain would miss the alternate day, but was sure to come the third day. On Sunday, if the patient remained perfectly quiet, the attack would be postponed until Monday, but if he took a walk or exercised in any way he was sure to suffer. The duration of the pain was always five or six hours. In the beginning of the trouble he suffered at times—usually in the morning—from dizziness or, to use his own words, he felt as if he were under the influence of liquor. The attacks always began in the left temporal region, with a feeling of numbness, and gradually extended toward the middle

[1] Read before the Medical and Chirurgical Faculty of Maryland, April 26, 1905.

line of the head with the point of worst suffering in the region of the supraorbital notch. The pain, at first slight, would grow more and more intense until, at the height of the paroxysm, the patient would walk the floor in agony. From this period of culmination, there would be a subsidence of the trouble, until it would disappear entirely. The patient consulted his family physician, who gave him anodynes, with no benefit. He then fell into the hands of two homœopathic physicians, neither of whom gave him any relief or an explanation of his pain. He then drifted around from one physician to another, and was in turn treated for stomach trouble, liver trouble, and nerve exhaustion. Thinking there might be some connection between the pain and his eyes, he saw an oculist, who told him that his glasses were correct. Two rhinologists were seen. One of them assured him that the pain had no connection with his nose and gave him such anodynes as he had not already taken. Finally, after several weeks of treatment, he suggested that the supraorbital nerve be cut. Just before consulting me the patient saw a prominent physician, who advised the injection of osmic acid into the nerve and the application of electricity over the affected area before having the nerve cut. He consulted me November 29, 1904. After hearing his history I felt that where so many had been unsuccessful I was doomed to failure also. From the history of the pain having followed "grip," from the periodicity, and from the fact that it was always localized over the left eye, I immediately thought of probable frontal sinus disease. I questioned him carefully as to a discharge into the nose or into the nasopharynx with a negative result. He denied having had any such symptom. Inquiries as to the condition of his stomach, liver, intestines, etc., revealed nothing worthy of note. His general health was good. He was wearing compound, hyperopic lenses, given him two years before by a very competent oculist, which corrected his astigmatism entirely. The edges of the lids showed a slight blepharitis. Inspection of the face gave no evidence of swelling in the affected area. Examination of the nasal fossæ showed on the right side a septal spur which did not interfere with respiration. In the left side, with the exception of an abnormal dryness of the mucous membrane, which I have several times noted in disease of the accessory cavities, there was nothing to throw any light on the mystery. After cocainizing, the most careful inspection failed to show pus anywhere in the nasal fossa. On being asked to localize the area of pain, the patient would place his finger in the middle line of the head just above the root of the nose and describe a semicircle outward almost to the external angle of the orbit. The pain never exceeded this area. Baffled by my examination of the nostrils, with the exception of the dry mucous membrane which added one link in the chain of evidence, I turned my attention to the supraorbital region. Transillumination of the two sinuses gave no information, one side being

as dark as the other, which was due, as I afterward discovered, to the thickness of the anterior wall of the cavities. Palpation and percussion over the left eye caused decided tenderness as contrasted with the opposite side. I then made a diagnosis of frontal sinus disease from the following points: 1. The pain followed what was undoubtedly an infectious disease. 2. The periodicity of the attacks quite common in frontal sinus disease. 3. The dryness of the nasal mucous membrane. 4. The extreme tenderness of the affected as contrasted with the sound side. The most valuable symptom was tenderness on percussion, which is almost pathognomonic of frontal sinus disease, while the group of symptoms seemed to point unerringly to the cavity as the cause of the headaches. Since he had had the pain for nearly a year, I advised an external or radical operation. The possibility of frontal sinus disease had not been suggested to the patient, and, not wishing to run the risk of possible deformity from an operation, unless it should be absolutely necessary, he decided to try the osmic acid treatment. The injections into the nerve were made, but did no permanent good. January 2, 1905, the patient came to my office in great pain and begged me to give him something to relieve him. He told me that he could see no relief in sight, and that he was fast becoming desperate. I prescribed for him, and again advised opening the sinus. He had suffered so long he was now ready to submit to any treatment that offered even a possibility of relief. On the afternoon of January 4th I operated at the Presbyterian Hospital, with the assistance of Drs. Carroll and Bray. Under ether anæsthesia an incision was made through the skin along the orbital margin, and the anterior bony wall chiselled through. This was perhaps three-quarters of an inch thick. On opening into the sinus there was a gush of pus. The mucous membrane was degenerated and covered with granulations. This was carefully removed with the curette, and the bone examined for a possible necrosis. The sinus was large, extending outward almost to the angle of the orbit and upward for at least an inch. After satisfying myself that no necrosis had occurred, I swabbed the cavity with chloride of zinc solution (20 per cent.), followed by alcohol and packed with iodoform gauze. The headaches have disappeared, and have not returned. The cavity is gradually filling up, which is the ideal result of the operation. Pus from the cavity showed staphylococcus pyogenes aureus in pure culture. The chief danger in these cases is the liability of necrosis, with consequent discharge of pus into the orbit and injury to the eyeball, or into the meninges, with the lighting up of a fatal meningitis. Logan Turner has called attention to the possibility of infection of the meninges through minute bloodvessels in the bone without any apparent necrosis.

The cardinal symptom of accessory cavity disease is the presence of pus in the nose. Its absence renders the diagnosis much

more difficult. M. Hajek, of Vienna, perhaps the greatest living authority on these diseases, says in his book (*Die Nebenhöhlen der Nase*, 1899) that in most cases of inflammatory frontal sinus disease pus is present in the nose. Exceptions to this rule are those rare cases in which the opening of the frontonasal canal into the sinus has a valve of mucous membrane, or there exists in the course of the canal an impermeable stricture. Such cases are doubtless exceedingly rare, and since there is a damming back of the secretion in the sinus, with eventual breaking through into the orbit and displacement of the eyeball, they do not fall into the hands of the rhinologist. The occurrence of such cases has been denied by Grünwald and Lichtwitz. Hajek himself has never seen such a case. And again he says that often the attention of the physician is attracted to the possibility of a frontal sinus disease on account of the intermittent, neuralgic character of a frontal sinus pain. He has often heard that a headache which comes on in attacks at the same time of the day is a typical neuralgia of the supraorbital nerve. Behind this typical, intermittent neuralgia how often has he found empyema of the frontal sinus. Particularly do the so-called influenza neuralgias of the trigeminus reveal themselves regularly as typical empyemata of the accessory cavities, especially of the frontal sinus. How easy it would be to heal many of these empyemata if the physician would only think of the possibility of frontal sinus disease and refer the patient to the rhinologist for immediate treatment, instead of after months of delay. Such words from Hajek must make an impression upon all of us.

It may be interesting to refer briefly to the pain of frontal sinus disease. It will be found in some cases that the patient will complain of pain only on alternate days. Such cases will simulate malaria, but treatment with quinine does no good. The pain usually comes on in the morning before eleven o'clock. The explanation is that during sleep there is no drainage through the frontonasal canal, and the sinus fills with pus. On arising in the morning there is a great pressure brought to bear on the opening of the canal, and this pressure produces pain until free drainage is established. I have never before seen a case where the pain came on in the middle of the day or in the afternoon. Such cases are certainly very unusual. In my case it is a question as to what became of the pus that was constantly being formed in the sinus. From the history of frontal sinus disease there should have been a profuse discharge of pus into the nose every other day. But repeated examinations, and the positive statements of the patient to the contrary, are certainly puzzling as to the escape of the pus. It does not seem possible that all, or even a large part of the pus, could have been absorbed into the system without causing decided symptoms, which the patient at no time had. I have not found an explanation of the afternoon attacks in frontal sinus disease.

I believe that many obscure headaches have their origin in some one or more of the accessory cavities of the nose, and that relief can only be obtained by intelligent examination and treatment of the same. The above case is presented as an illustration of a headache which could not possibly have been cured without an operation.

PERSONAL EXPERIENCES WITH EMPYEMATA OF THE FRONTAL SINUS.[1]

By George L. Richards, M.D.,
otologist and laryngologist of fall river union hospital, fall river, mass.

At the present moment the therapy of frontal sinus affections is one of the much discussed and still unsettled problems in rhinology. During the last ten years various methods have been proposed, enthusiastically commended, and then allowed to drop more or less into disuse. These have varied from the simplest to the extremest methods of operation within the nasal canal, and from simple opening and prompt closure externally to the present much-advocated, very radical procedure of Professor Killian. As a contribution to the general subject the writer begs leave to offer for the consideration of this Association his own personal experiences in empyemata of the frontal sinus, together with some deductions from these experiences, and some observations as to what he regards as the probable future trend of operative procedure in this locality.

While the number of cases presented is not large, and while it is unsafe to reason too much from too limited experience, it is hoped that the deductions may have some value. The cases are reported somewhat in detail, the exact operative procedure being described rather than named after anybody in particular.

In his address at Atlantic City before the American Medical Association, 1904, Professor Logan Turner stated that what was really needed at the present time was an accurate report of the chronicity of the cases, the number of cases affected, the surgical method employed in operating, the treatment of the wound, and the results obtained as regards cure, relapse, the amount of disfigurement produced, and the fatal issue. As a contribution to such a qualified investigation I offer the present cases.

At the outset a certain amount of differentiation of cases must be made. I discard entirely that large group of acute inflammations of the frontal sinus which occur perhaps more and more often as accompaniments or sequelæ of grip, which may or may not be

[1] Presented as candidate's thesis, American Laryngological Association, Atlantic City meeting, 1905

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VOL. CXXX., No. 5. NOVEMBER, 1905. No. 404.

THE

AMERICAN JOURNAL

OF THE

MEDICAL SCIENCES

Published Monthly.

EDITED BY FRANCIS R. PACKARD, M.D.

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PHILADELPHIA AND NEW YORK:

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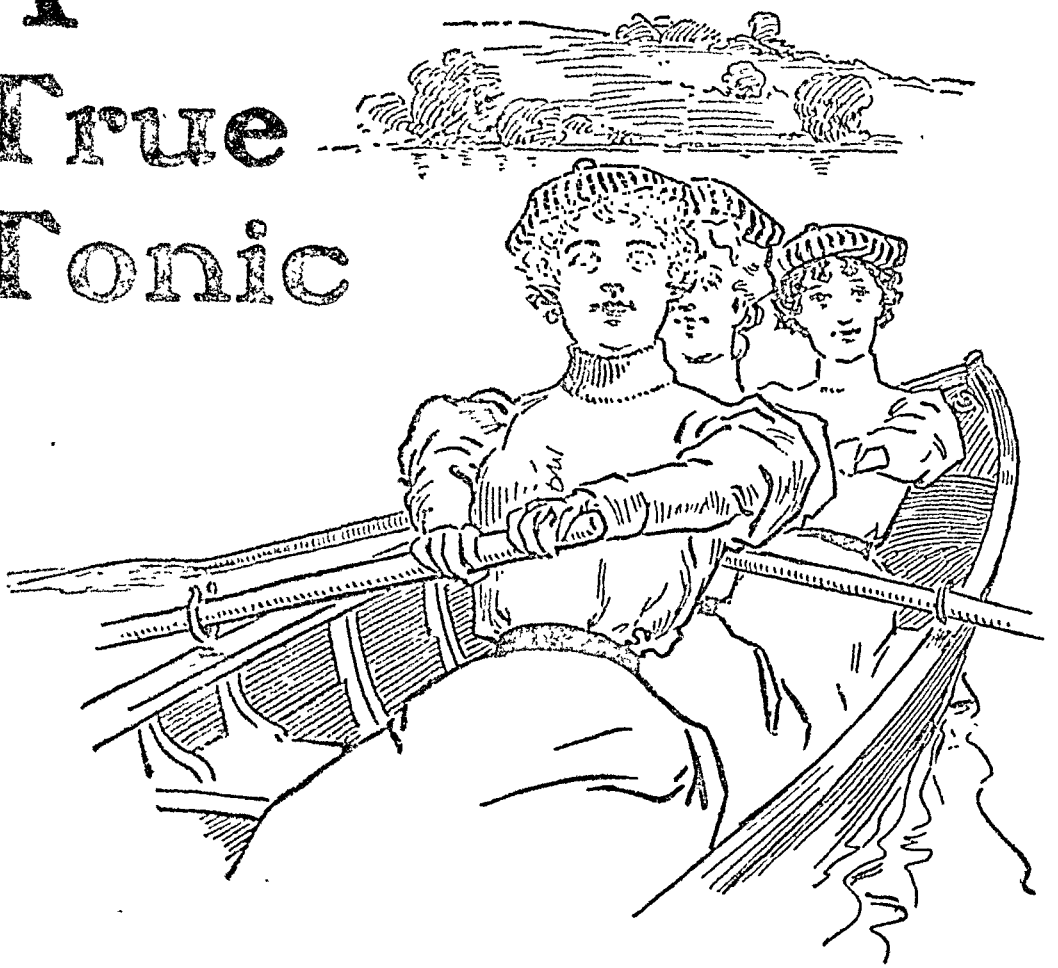
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accompanied with purulent exudate, and which recover promptly under simple nasal treatment, sometimes with, sometimes without, removal of the anterior end of the middle turbinate.

The chronic cases of empyemata may also be divided into two classes: first, those in which the reasonable persistence of intranasal therapeutic measures has brought about success; and, second, those which, after the persistence for a reasonable time of such intranasal therapeutic measures as we have at our command, are still uncured or not sufficiently improved, and in which an external operation seems imperatively demanded.

Of the first group of cases the following four are examples:

CASE I. *Chronic inflammation of the left frontal sinus.*—Retired sea captain, aged seventy years. This case was referred to me on account of a great deal of mucopurulent discharge of long standing from the left nostril in a man of far from robust health and subject to attacks of heart difficulty.

There were numerous polypi in the left nostril, which were removed, leaving behind a steady discharge of foul pus in the middle meatus, with bare bone in the region of the frontal sinus outlet. Exploratory incision was made into the antrum, but there was no pus. I was able to pass a cannula directly into the frontal sinus after cutting away some diseased tissue in the region of the outlet with the Grünwald cutting forceps. The middle turbinate as a whole was not removed. The quantity of pus diminished, the odor gradually grew less, and the patient's general condition better. The sinus was syringed out every other day with 1: 8000 corrosive sublimate. On one occasion this syringing made him dizzy, requiring an hypodermic of strychnine and morphine before he was able to go home.

The discharge steadily diminished, and on April 1, 1902, he having come under observation on November 26, 1901, there was absolutely no pus discharge and he felt perfectly well. He was seen occasionally from this time until December 10, 1903, and at no time was there any sign of discharge from the nose or any trouble in the sinus.

This case shows what can be done under apparently difficult conditions even with a foul purulent discharge. I was not allowed from start to finish to do anything but the mildest kind of operations such as could be done with a snare or a few strokes of a cutting forceps, yet, to all intents and purposes, this patient was as perfectly cured by treatment within the nose as he would have been by any external operation. Three years have now elapsed without any return of the trouble. I have recently seen him and he remains in good health. There has been no nasal discharge since the cessation of treatment.

CASE II. *Subacute inflammation of the right frontal sinus.*—Mrs. W. H., aged twenty-six years. First seen January 28, 1903. Had

history of pain in the head following grip three months ago, with a discharge from the right nostril for five weeks. Complained of pain in the right side of the head and soreness above the inner angle of the eye. Pain was very much worse when leaning over and of a throbbing character. There was pus in the right nostril. The middle turbinate was crowded against the septum and against the bulla so that the space for drainage was very poor. Could not probe the sinus. Provisional diagnosis of subacute empyema of the right frontal sinus.

The next day removed anterior portion of the right middle turbinate in order to get better drainage. The pain over the right eye soon diminished, and in a few days she was quite comfortable. The pus discharge continued, gradually diminishing. I was able to carry the cannula up into the sinus and syringe it out, but only with the greatest difficulty.

Two months later there was still discharge from the nose in spite of daily treatment. It was difficult to wash out the sinus very successfully, and I thought the only cure would be by means of an external operation, which I advised but which was refused. A few days after this I removed the rest of the middle turbinate and was able to get farther up toward the opening to the frontal sinus, but its washing out was only fairly successful, owing to a very narrow passage.

As, however, the patient refused any external operation and the discharge slightly lessened, and as the infundibular opening was gradually enlarged so that various solutions could be carried up into the sinus, the idea of an external operation was given up, and on May 19th there was apparently a cure of the sinus condition and the case was dismissed. Six months later there was no apparent difference on transillumination between the two sinuses, and no sign of trouble in the nose. At the present time, a year and a half later, there has been no further trouble. Her husband, however, reported to me that following an acute cold this winter there was a little sinus pain, which soon disappeared.

In this particular instance, although there has been an apparent cure by intranasal methods, I think it would have been better surgery to have opened externally, as I think there is a likelihood of a return of the trouble after an attack of grip or any acute infective disorder.

CASE III. *Chronic inflammation of the right frontal sinus, with acute exacerbation.*—Dr. E. E. R., aged forty-five years. Has a history of several times in the past having what was called severe neuralgic pains of the right side of the head, right eye, and region of the right sinus. At one time gave up his work for nearly an entire year, not having any relief at all until after three or four months in the summer in the dry air of the highlands of New Hampshire, after which he was free from this pain for several years. While travelling in a

sleeping car recently, March 12, 1904, was taken with severe pain in the region of the right frontal sinus. Had no relief from treatment. Had to give up his work, that of an active physician.

Examination of the nose showed the right middle turbinate engorged, so much so that adrenalin did not blanch it. No pus was visible. The right middle turbinate was at once removed, giving some relief to the severe pain. Could enter the sinus with a probe. The pain greatly diminished until, four days after seeing him, was able to wash out the right frontal sinus through a cannula, removing a considerable quantity of mucopus, with immediate relief of all the symptoms. Washing was continued, and at the end of a week he was well and has continued so to the present time.

This was one of those cases of severe involvement of the frontal sinus in which, if the removal of the middle turbinate can be promptly done, the sinus drained, and relief of the engorgement brought about, a cure will be effected without further operative procedure.

CASE IV. *Subacute inflammation of right frontal sinus.*—A. S., aged forty-four years, designer, came on April 12, 1904, complaining of great pain over the right eye, which he thought was due to trouble with his glasses. The oculist reported that they were all right. All nasal structures on that side congested. Treatment, nasal irrigation.

April 14th. Pain has been almost continuous. Unable to sleep. I at once removed the middle turbinate on the right side. After the operation was able to pass a curved cannula directly into the right frontal sinus, washing out some thick purulent debris.

15th. In spite of the pain which followed the operation and the necessity of wearing a cotton tampon, has had a better night than for weeks. Washed out the frontal again. April 16th, washed out the frontal sinus. No further pain over the eye.

19th. Tried to go to his work, but gave it up and went to bed. Some reaction evidently following the operation. Washed out the sinus, but no pus. From this time on the nasal discharge entirely ceased. He had an attack of acute rheumatism, for which he went into the hospital, but there was no pain in the region of the frontal sinus, and when seen the last time, on May 12th, there was no discharge and he was perfectly well.

This was a case of subacute inflammation of the frontal sinus with an acute exacerbation in which the prompt establishment of free drainage cured the case. It is also one of those cases which, improperly diagnosed, tends to go on to a chronic condition with constant pus discharge.

The following group of cases belongs to Class II.—viz., those which remain uncured or insufficiently improved after faithful use of intranasal measures, or in which, for urgent or other reasons, intranasal measures are manifestly not indicated.

CASE V.—Mrs. G., aged thirty-five years, seen October 17, 1899, on account of severe headaches. Examination showed pus in each nostril with polypi. The polypi were removed and then each middle turbinate.

December 15, 1899, the right frontal was opened externally at the inner angle of the eye underneath the supraorbital ridge. There was fluid pus but no granulations. The opening was extended down into the nose; the external wound was treated as an open wound for two weeks and then allowed to close, at which time the probe passed easily into the nose from the right sinus. There was improvement of the pain but the pus discharge continued, and later complaint was made of pain over the left sinus. This was then opened in the same manner and the right one reopened. The two did not communicate. The opening into each nose was made larger with good-sized curettes; iodoform gauze wicks passed through, followed in a few days by good-sized rubber drainage tubes passed clear into the nose and retained for four weeks. The wound was then allowed to close externally. The patient's general condition was much improved, although some pus discharge continued, varying more or less in amount.

A few months later the sinuses were again operated on and larger drainage tubes kept in for several weeks. There was improvement as before, but the pain returned again some months later and the discharge, which had never stopped, became worse, so another operation was done July 27, 1900. This time the incisions were made above the supraorbital ridge. It was found that the right sinus, except at its most dependent portion, was completely closed with osseous growth as the result of the former operation, but there was pus in the left sinus.

For something over a year the condition was not very troublesome, but November 8, 1901, on account of throbbing pain and some pus discharge, the sinuses were each opened at the most dependent portion of the inner angle of each eye, found nearly closed with bone, the trouble being mostly at this time in the cells in the dependent portion of the sinus. All of the ethmoidal cells which could be reached were curetted and destroyed. The rubber tubes were inserted and retained five days, followed by silver tubes worn for three weeks. She was seen occasionally for some months, then I observed that there was an empyema of the left antrum. This was due to infection, as on previous occasions both antra had been found free from pus.

On July 23, 1902, a radical operation for the empyema of the antrum was performed through the canine fossa, followed by rapid healing; the antrum at the time of the operation containing considerable granular debris. After this was done she was not seen for a year, during which time she became pregnant and had a normal labor. At this time nothing special was found

in the nose, although she still claimed to have more or less pus discharge.

On July 12, 1904, she was examined. There was a little mucopus in the nose, but she was practically well and in good health, all the pain having disappeared.

On April 11, 1905, re-examined; both frontal sinuses apparently obliterated. There was a little dried scabbing in the floor of the nose, due apparently to atrophic conditions. Probe detected bare bone in the right sphenoid; left sphenoid could not find the opening. The patient's general condition good. This case is a cure so far as the empyemata of the frontal sinuses are concerned. It is doubtful whether all of the ethmoidal cells are in a healthy condition.

Criticism. This was a case of extensive ethmoiditis with involvement of each sinus, and later infection of the left antrum from the sinus. My early operations were without doubt incomplete. Not having been followed by complete obliteration of the sinuses, there was continual reinfection until, as a result of the many operations, a final cure was effected. The early operations were too timid and the openings into the nose were not large enough nor the infected ethmoidal cells thoroughly destroyed. The sinuses were not extensive in area, and had complete obliteration, by packing, been attempted, a cure would no doubt have been effected much earlier.

CASE VI.—Mr. F. W. N., aged thirty-two years, milkman, resident of Martha's Vineyard. Seen in November and December, 1898, for polypi and pus discharge lasting a long time. The polypi were removed with so much improvement that he did not present himself again for a year, at which time the pus was still present and the polypi had recurred.

The middle turbinate was removed, and under ether January 12, 1900, I made an opening into the right frontal sinus at the inner angle of the eye, making an incision in the ridge from the supraorbital foramen to the nasal process of the superior maxilla. The opening into the sinus was made at about the mediate point of this incision, around the inner angle of the eye. The sinus was filled with free-bleeding granulation tissue, all of which was curetted out, and the opening enlarged so as to get free drainage into the nose. A wick of iodoform gauze was carried down into the nose, followed with rubber drainage tubes, and at the end of two weeks, the sinus being apparently in good condition, the external opening was allowed to close, and the patient returned home.

He was seen once or twice afterward and then not again for something over a year, when he reported that there was some pus in the right antrum, easily blown out when the right cheek was uppermost. He was not seen again for a year when, January 7, 1902, he reported that there was some discharge from the nose in winter. Examination showed no pus presenting at the frontonasal

canal, but a little bare bone could be felt with the probe, and there was no doubt but that the sinus was not perfectly healed.

April 10, 1905. This patient has just been re-examined. There is no sign whatever of pus in the nose, and he states that there has been no discharge from the frontal sinus so far as he knows for five years. Two or three times in the winter he has had a little sense of pain in the right maxillary sinus, followed by a discharge of mucus and mucopus, quickly stopping. This would apparently be due to a mild attack of inflammation of the maxillary sinus to which reference has already been made as having occurred in the past. Transillumination of both frontals was done, the right frontal showing almost entire obliteration, the left frontal extending high up on to the forehead, with two or three septa plainly visible. Transillumination of the two antra showed each one perfectly free, so that at the present time a perfect cure of all the accessory sinus trouble has apparently occurred.

Criticism. This was another one of the cases in which, viewed in the light of later events, I think that complete obliteration of the sinus should have been attempted at the original operation, and that presumptively the small amount of pus remaining, with the possibility of colds and inflammatory action in the nose, very likely infected the maxillary sinus. As, however, the patient was dependent upon his daily labor for existence, and as he lived where he was practically inaccessible, it seemed as though the less radical operation would probably answer, and this has in fact occurred, as with the lapse of time a perfect cure has resulted.

CASE VII.—L. J., student, first seen April 5, 1899, when sixteen years of age. Brought on account of fear that he had tuberculosis. Had had a cough for ten years. One sister had died of tuberculosis and he had spent the previous two years in Colorado and New Mexico. Was unable to find any tubercle bacilli in the lungs, but micrococcus tetragenus aureus and streptococci were abundant. Under treatment the chest conditions improved. During this time he had an acute otitis media in both ears, relieved by incision. Was under my care at intervals for a year, during which time I removed a few polypi from the right nostril, and a year later, June 27, 1900, recorded that the nose was running a good deal. There was some discharge apparently from the left frontal sinus. Transillumination did not show any very well-defined shadow. He was relieved by washing out the nose with hot Seiler's solution, which treatment he continued on his own account. About this time he entered Brown University, completing his course there in about three years.

From June 27, 1900, to May 29, 1903, I have no record in regard to him, though I saw him occasionally. The lung conditions had very much improved. At this time he came complaining of discharge from the nose, which he said had continued more or less for

several years. Examination showed pus in the nostril, and exploratory puncture demonstrated it in each maxillary antrum. These were washed out at intervals and numerous polypi removed. The right middle turbinate was also removed. Diagnosis of empyema of frontal as well as maxillary sinuses, and operation advised.

On June 17, 1903, he was operated on in the hospital, the left sinus, the left antrum, the right sinus, and the right antrum all opened. Pus and diseased mucous membrane and polypoid degenerative material was found in each cavity, the antra being practically filled. The frontal sinuses were opened at the inner angle of the eye, underneath the supraorbital ridge, the greater part of the front wall of each being cut away, while the frontonasal canals were each enlarged sufficiently to take a large drainage tube, and the adjacent ethmoid cells curetted. The entire mucous membrane of each sinus was curetted out. The sinuses did not communicate and were of ordinary size both as regards extension upward and outward. A large drainage tube was carried down into the nose from each sinus. Each maxillary antrum was opened through the canine fossa; all the diseased mucous membrane curetted away, and a large counter opening made underneath the floor of the nose. All the cavities were packed with gauze.

The after-treatment was uneventful so far as the maxillary sinuses were concerned. The gauze was kept in fifteen days and then not replaced. A gutta-percha obturator was kept in each antral opening for about two weeks afterward. Then they were allowed to close. There has been no further trouble from them. The opening underneath the inferior turbinate has continued to the present time.

The frontal sinuses at this time were granulating in a healthy way and were being allowed to close in, gauze dressings being retained, the tubes having been removed about ten days after the operation. The gauze put in was saturated with balsam of Peru. At this time I was taken ill with typhoid fever and did not see the case again for nearly three months, during which time he was under the care of a colleague, who allowed the openings to close up. When I saw the patient they had broken out again at the surface, and the right one, he said, had never entirely healed. Both were curetted. The left one had a very little superficial pus. The right one showed some soft bone under the supraorbital ridge and some thick pus with some polypoid tissue at the nasal outlet. Gauze drains were replaced.

October 24th, three weeks after, both sides were closed; the right nostril was discharging a little pus, the left none. November 18th, a month later, there was a little accumulation of pus from the right sinus. December 24th, the left middle turbinal, which had not been previously removed, was taken out and a polypus from the region of the right frontonasal canal.

February 2, 1904, the right sinus broke out again with slight pus discharge, but the probe followed only a narrow track. Did not find much of a cavity. Opening into the nose still patulous. Curetted.

May 9, 1904, reported that he had some mucus discharge from the nose, but did not find any source from which it came. May 28th found several small polypi in the right infundibulum and just a suggestion of pus. Curetted a little. Some anterior ethmoidal cells not wholly cured.

July 15, 1904, pocketing of pus had again occurred on the right side. On opening was able to pass a probe through a small opening down into the nose. The left sinus remained completely closed. Remnants of the right sinus were very small. Curetted thoroughly. Washed down into the nose with pyoktanin solution, and allowed to close again in the hope that complete obliteration would this time take place.

At present, September, 1905, the sinuses are apparently well, but there is occasionally a slight mucoid discharge from the ethmoid region, for which I have recently removed several of the anterior ethmoid cells, and regard him as practically well. No appreciable scar remains as the result of the operative measures.

Criticism. As the sinuses in this case did not extend upward or outward beyond the ordinary average limits, and as the external wound could have been kept open sufficiently long for the obliteration method, I think this case would have done better if, at the time of the original operation, the opening down into the nose had been made larger, the anterior ethmoidal cells curetted more vigorously, the bone opening continued lower down, cutting away a portion of the nasal process of the superior maxillary, and then the external wound maintained by gauze packing until complete obliteration had taken place. This was in my mind to do, but unfortunately my own illness prevented, nor had I at that time become so thoroughly convinced as at present of the absolute necessity of obliterating the sinus in order to prevent a recurrence. Nevertheless I believe that a cure has finally occurred.

CASE VIII.—G. F., aged twenty-six years. First seen June 21, 1899, having developed a frontal sinus trouble as the result of trauma, he having been injured in a railroad accident. The exact nature of the trouble was not discovered for some time, the case having first been reported to me as a case of atrophic rhinitis with a foul discharge. Was treated by the usual washing methods for a while, with opening through the canine fossa made with motor drill. The pus continuing in spite of pretty free drainage from the antrum, the question of frontal sinus trouble came up. He was seen by Dr. Leland, who thought the frontal sinus involved and advised an operation on the sinus. He was complaining a good deal of headache, which seemed to be due to the frontal sinus trouble.

On June 19, 1900, the right frontal sinus was opened at the inner angle of the eye. Pus was present but not in large amount. There was no odor and very little granulation tissue. Sinus area extended up about one-half inch and outward one and one-half to one and three-quarters inches from this point. The opening down into the nose was enlarged and a large-sized piece of gauze wicking put in. The right maxillary antrum was then freely opened through the canine fossa above the place previously opened and filled with gauze. The frontal sinus opening was packed with gauze two or three weeks, the gauze being followed by a small gold tube, which was worn for two or three months. Its removal was followed by complete closing of the frontal sinus opening, the sinus being completely obliterated.

This was in July, 1900, and there has never been any reopening of the frontal sinus, nearly five years having now elapsed. There has been occasionally a little bit of mucopus in the antrum, and he has insisted all these years on maintaining a small opening from the mouth into the antrum, which he washes out once or twice a week. I have not been able for some three years to demonstrate any pus in the antrum, but he feels better if he can wash out the antrum occasionally, and now and then imagines that he has a little pus. Practically, however, the case has remained perfectly well for the last four years, and the frontal sinus has never broken out.

In this instance, with a comparatively small sinus and rapid formation of granulation tissue after the operation the gold tube seems to have worked as well in maintaining the opening and to have been rather more comfortable to wear than the gauze. One reason for the good result may have been due to the fact that infection was secondary to antrum trouble and there was not extensive involvement of neighboring ethmoidal cells.

At the present time there is no appreciable scar, close search being required to determine the site of the operation.

CASE IX.—O. M., aged seventeen years, spinner. First seen February 11, 1902, at which time she complained of a nasal discharge and pain in the region of the right frontal sinus. Gave a history of the discharge from the right side of the nose for one year with pain in the right side over the right eye. Examination showed pus in the right nostril, coming from high up in the nose. Probe detected bare bone in the infundibular region. Transillumination was negative. Probable diagnosis of empyema of the frontal sinus made.

February 15th removed the entire middle turbinate on the right side. Diagnosis then positive. Could pass the cannula into the sinus and syringe out the pus.

On March 5th was operated on at hospital for empyema. Incision made beneath the supraorbital ridge at the inner angle of the eye. Periosteum laid bare. Bone darker than normal; thin over the

sinus wall; opened; pus and granulations; no odor. Enlarged the opening; removed the granulations. Enlarged the opening into the nose so that a fair-sized rubber drainage tube was easily passed into the nose. There were no symptoms pointing to antral trouble, so the antrum was not opened. There was no connection between the two sinuses. The area of the frontal sinus was not excessive, not going very high above or outwardly.

Treatment in the hospital uneventful. Occasional slight temperature rise for a few days. Dressing changed on the third day for the first time. New tube put in on the seventh day. Left the hospital on the twelfth day, at which time the drainage tube was still in and the pus discharge very slight. The tube was removed a few days later, followed by gauze in the external opening. Pus discharge almost stopped, but not entirely. From this time until May 3d the nose and the external wound were washed out every other day, the external wound being practically closed in.

As there was still some pus from the nose, I punctured the right antrum and found pus, and on May 7th, two months after the first operation, again put her in the hospital and did a radical antrum operation. There was some pus but not much degeneration of the mucous membrane. A large opening was made into the canine fossa with a counteropening underneath the anterior end of the inferior turbinate. This was packed with gauze. The frontal sinus was again curetted. At the external wound there was no pus, but there was more or less rather poor granulation tissue. The opening was packed with gauze. Slight rise of temperature after the operation, but hospital record practically negative.

On May 15th the communication between the sinus and the nose had closed. There was some pus discharge from the frontal sinus wound around the gauze, but it was granulating and filling in. May 20th there was no pus in the nose from the frontal sinus. On May 24th the frontal sinus wound was still discharging a little pus. She was given chloroform in the office, and some low form of granulation tissue recuretted, and instead of the gauze I put in a curved silver tube, carrying it down into the nose preparatory to letting the cavity close.

The antrum wound had had the gauze dressing continued and had done perfectly well. From May 27th to June 5th there was no apparent pus from either cavity, and the case, being apparently cured, was discharged.

On August 28th, three months later, she returned with the opening in the frontal sinus broken out, small accumulation of pus causing some œdema in this region. Under chloroform skin incision was made, the opening down into the nose found with the probe, and the cavity packed with gauze. The dressings were then done every other day, and on September 9th there was apparently complete healing, and the case was again dismissed.

Two months later she was again seen. There had been no trouble. Not seen again until ten months later, June 2, 1903, when she returned with a small area of pus presenting from the right sinus. Chloroform was given and the pus evacuated. It seemed to be in the surface only. The sinus was apparently obliterated and the probe did not go any distance. A gauze drain was put in, and on June 9th there was apparently complete healing. Case has been well ever since and there is no deformity.

Criticism. In this case the antrum was probably infected from the frontal sinus, and the diagnosis of the antrum trouble should have been made coincidently with the diagnosis of the trouble with the frontal, and both operations should have been done the first time providing there had been pus in the antrum.

With reference to the treatment of the frontal, in which there was a recurrence of trouble in the granulating area on three different times, this should have been treated by the method of absolute obliteration with gauze drainage from the start, and the opening into the nose should have been made sufficiently large so there would be no likelihood of that closing during the period of granulating above. So far as can be seen, a perfect cure resulted with complete obliteration of the sinus, but the time required seems to me to have been longer than it should have been.

CASE X.—Mrs. E. S., aged forty-six years. Came on account of catarrh on April 10, 1903. Examination of the nose showed numerous small polypi around the anterior end of each middle turbinate. These were removed, and on April 15th the right inferior turbinate was removed. She suffered greatly from inability to sleep. April 25th several polypi had come down from the ethmoid region where the turbinate was snared off.

May 15th some pus from the frontal sinus. Washed out without difficulty. Probable diagnosis of old empyema of frontal. The sinus was washed out for a number of days and the infundibular region curetted of polypoid tissue. The pus lessened in amount but continued. The antrum was opened exploratorily but no pus found.

On June 17th the right sphenoid sinus was opened and cleaned out with a curette. The walls were rough, but no apparent pus. On June 24th the right frontal sinus was opened under ether. It was small in area with soft anterior walls, filled with pulpy mucous membrane, which was removed entire, the opening into the nose enlarged, large drainage tube inserted, and the sinus cavity packed with gauze.

The after history is uneventful with the exception that on July 7th, two weeks after the operation, she complained of seeing double. The affected eye could be rotated in every direction, and I was not conscious at the time of operation of injuring the pulley of the superior oblique. The double vision gradually lessened, and on

November 28th had entirely disappeared and she was perfectly well. This case has been recently seen and there has been no recurrence. The opposite side, which had shown some polypi, has ceased to trouble her, and there is no pus discharge or any trouble whatever.

Criticism. In this case the anterior bony wall of the antrum was so soft as to break away easily under the use of a curette, hence extraordinary care should have been taken when in the region of the pulley of the superior oblique. This is the only case of double vision which I have seen following frontal sinus trouble.

CASE XI.—W. G., aged twenty-nine years. First seen March 22, 1903, on account of pus in the right nose and right ear. Said to have followed an attack of grip. There was an otitis media purulenta with perforation.

The right nostril was filled with polypi and there was a constant pus discharge. Exploratory puncture into the antrum showed the presence of pus, and the case was treated by washing methods. The polypi were removed, after which it was possible to probe the frontal sinus and wash out through a cannula. The frontal sinus was irrigated daily with a great deal of care, and this method of treatment carried on an entire month without much if any improvement, except temporarily. Radical operation was then advised.

The right frontal sinus was opened underneath the supraorbital ridge on the right side. Pus immediately welled out in considerable quantity. The opening was enlarged. The sinus itself was very large, extending clear over to the left side as far as directly above the supraorbital foramen on the left side, and quite a distance up on the forehead. I thought at first there was a communication between the right and left sinuses, and was on the point of opening the left sinus but did not do so. Cleaned out considerable polypoid tissue in addition to the pus. Opening into the nose enlarged and drainage tube inserted.

A radical operation was then done on the antrum. It was found partly filled with large polypoid masses, some of them of considerable size. The usual counteropening was then made into the nose. He was kept in the hospital two or three weeks. Had more or less pain and swelling of the eye.

At this time I was away for three weeks, when he was under the care of another physician. Both cavities had been washed out daily. This was on May 16th. Examination showed the antrum apparently nearly healed. The frontal sinus had a silver tube in it. Opening down into the nose still present and of fair size. Examination showed a very large sinus going away over the left side.

Decided to open the left sinus for fear that the two sinuses communicated, and if this were not done some pus pocket might be left which would cause trouble in the final healing. On May 17th the left sinus was opened under ether. Could not find any communi-

cation whatever with the right one. This sinus was very small and quite limited in extent, communicating freely with the left nostril. The mucous membrane was pulpy, detached at one place, and on pulling came away entire. Made a good-sized opening into the nose. This sinus promptly healed by the obliteration method, and has caused no trouble since. The other sinus continued to discharge a little. The patient, however, went back to work. Felt perfectly well. Gold tube was kept at the external angle of the wound, and he was seen by me once or twice a week.

October 24th, had been at work through the summer, but reported at this time on account of some pain in the left eye. The opening between the two divisions of the sinus had closed, and pus had accumulated in the left side. This portion of the sinus was curetted as thoroughly as possible under ether, and gauze drain put in.

For the next two or three months there was slight discharge. Various things were used for syringing and the discharge became small in amount. Still there was always a little. During the following spring solutions of pyoktanin and boric acid were used to wash out the cavity, and under this the discharge became almost nothing. The opening into the nose continued, and on January 30th, at the meeting of the Eastern Section of the American Laryngological, Rhinological, and Otological Society, he was seen in consultation with Dr. C. G. Coakley, who thought that permanent cure would not be reached until every particle of mucous membrane was removed, which could be done best, in his opinion, by taking off the entire front plate of the sinus. It might be attained by opening up through the already obliterated left sinus. As, however, the patient was in good condition, able to labor, but unable to afford the time to give up work, it seemed to me doubtful if any further operation was then advisable.

From this time for the following four or five months the discharge was almost nil. Four, five, or six days would elapse without any, and then there would be a little at the angle of the eye. Examination with the probe on June 15th showed the sinus to be apparently closed on the left side, but there was still some bare bone to be filled in the region of the supraorbital foramen on the right, and the probe passed readily down into the nose. Curetted toward the outer portion of the cavity to the right and a drop or two of pus came out. Removed a couple of polypi from the nose. At this time I used the pyoktanin in powder form, blowing it into the cavity.

From July, 1904, to December, 1904, he was seen by me only three times. On December 14th he reported there had been no pus discharge for an entire week. The probe did not hit any bare bone. There was almost no trouble in the nose. Up to the present time an occasional polypus had formed at the nasal outlet of the frontal sinus opening.

There has been no discharge of any account since December. Recent examination (September, 1905) shows small external opening into right sinus, which is to be closed by suture. The portion of the sinus leading to the left side obliterated, or else, which is more probable, division wall grown up so there is no communication. Right sinus much smaller. Opening into the nose easily found with a probe. No discharge of any amount and no polypi.

Criticism. This is one of the cases where, in the light of subsequent history, either an entire resection of the front wall of the sinus ought to have been done at the time of the first operation, or else at the time the second operation of the left frontal sinus was done, this should have been opened clear up so as to throw the cavity of the right and left into one, thereby giving better access to the cavity of the left sinus. The entire treatment consisted of drainage only, and as I was unable to remove the entire mucous membrane at any one operation, the case can hardly be called an absolute cure, even though for the last year or year and a half there are practically no symptoms. Should he have an acute infection or an attack of grip or something of that sort, I think this sinus is liable to be reinfected, and although I have an apparent cure, I can hardly report this case as an absolute cure. At the same time the patient, who was very much run down physically when first seen, is strong and well and hearty, and has had no pain or discomfort from the sinus for more than a year.

CASE XII.—Dr. C. First seen January 27, 1904, with a history of illness for several weeks and a left-side nasal discharge, for which his physician wished me consulted. Examination showed creamy pus, coming apparently from the left frontal sinus, perhaps from the antrum as well. Transillumination showed extreme darkness as compared with the opposite side. There was some darkening over the left antrum. Exploratory puncture, however, showed the antrum to be clear. Nose was washed out for several days, giving some relief, and February 8th he had an acute otitis media, for which the drum was incised and gauze drainage used. There was some mastoid tenderness, but recovery from the ear trouble occurred without real trouble with the mastoid.

The sinus was washed out with pyoktanin solution for one or two weeks. Soon after this pus appeared to come from the right frontal sinus as well as the left. Both middle turbinates were then removed. The discharge from the sinuses diminished but did not disappear.

April 15, 1904, decided to do a radical operation. Opened the left frontal sinus by curved incision between the root of the nose and the inner line of the eyebrow. There was no fluid pus but a pulpy condition of the entire mucous membrane. Sinus in area about the average, not running up on the forehead to any great distance, though farther on the right side than the left. The entire mucous membrane was pulpy, thickened, and everywhere diseased. Re-

moved it entire with thorough curetting. Underneath the membrane the bone seemed hard and sound. Just above the root of the nose was a small opening about 3 mm. in diameter communicating with the opposite sinus, and through which it is probable that the left sinus infected the right, although there seemed to be no direct communication between the two membranes. After thorough curetting of the interior, the probe was passed down into the nose and this opening enlarged to carry good-sized rubber drainage tube. It was fastened above and below and the cavity packed with gauze.

The right sinus was then opened in the same way, the front wall, however, was very much thinner on this side, and the periosteotome easily penetrated the sinus when engaged in stripping the periosteum. On incision through the mucous membrane of the sinus, fluid pus was found to fill the sinus. This sinus extended quite a way over the eye and also above much farther than on the left side. The entire mucous membrane was curetted out and removed, and the opening down into the nose enlarged as before and drainage tube inserted. The bony opening between the two sides was then enlarged and gauze placed between. Each cavity filled with small strips of iodoform gauze.

The after-history of this case is uneventful. It was kept packed with iodoform gauze, soaked at intervals in balsam of Peru, and changed every day or every other day, and on August 12, 1904, the case was dismissed, complete healing having taken place. The point which filled up last, and which was in the end the most annoying, was the small bony septum between the two sides, to which the granulations did not seem to attach very well, probably because of its insufficient blood supply, and I regretted that I had not enlarged the opening between the two sinuses more. This is the only criticism I have to offer on this case. Healing up to the present time has been complete, and the patient, who had suffered from various vague head symptoms for many years, is better than he ever remembers himself to have been before.

CASE XIII.—M. B., aged twenty years. First seen with nasal symptoms on March 2, 1900, at which time some pus presented from underneath the left middle turbinate and two small polypi were removed from this space. Probe was carried up into the frontal sinus. Transillumination showed opaque left antral region as against lightness on the opposite side. Pus was very apparent in the middle meatus. Diagnosis made of pus in the antrum, perhaps in the frontal.

March 7th was operated on under ether, a radical antrum operation being done. The antrum contained pus and soft, pulpy mucous membrane. This was entirely removed and the usual counteropening made into the floor of the nose. Frontal sinus not opened. The healing of the antrum was uneventful, but after this occurred

there was some pus still apparent in the nose, which did not disappear after syringing, and on April 23, 1900, the frontal sinus was opened underneath the supraorbital ridge at the inner angle of the eye. The bone was very thick. Pus abundant and very foul. Rubber drainage tube carried down into the nose and maintained for some time.

On May 14th the cavity had been allowed to close, trusting to nasal drainage. On June 8, 1900, there was a drop or two of pus exuding through the wound, so that a small curette was put in. Cavity curetted and packed with gauze. Healing of the external wound promptly took place, and I did not see him for some time, but on August 7, 1900, he complained of pain. Could not find any sign of pus in the external wound or nose. Seven days later, however, there was some sign of pus externally. This was curetted. Gold tube placed in for drainage and the probe carried down into the nose, readily finding the remnant of the opening made for the tube. Tube was maintained as long as possible, but the latter part of August the opening had closed, and September 19th, the case showing a tendency to continually reopen, he was again etherized. The frontal sinus recuretted thoroughly. Some soft bone found. The opening into the nose reopened and enlarged and the whole packed with gauze. By October 25th the opening had nearly closed, gauze having been kept in continuously, and the opening was allowed to heal up.

On November 8, 1900, the wound broke out again. There was a small amount of pus present. The whole area of softened bone again curetted. Small piece of gauze inserted. November 17th and 22d drop or two of pus found in each instance, and the latter time was able to carry the probe clear down into the nose. Syringed some peroxide through. The opening again allowed to close.

December 3d pus again found in considerable amount in the upper portion of the frontal sinus. Curetted again. Piece of gauze put in. December 10, 1900, under cocaine, curetted the upper portion of the frontal sinus over against the dividing line from its fellow of the opposite side. Put in gauze drain again. Washed out every day for the next month. December 29th no pus found in the sinus. January 15, 1901, a drop or two of pus showed in the angle of the wound. Frontal was again opened with the knife. On February 11, 1901, there was apparently complete cure.

For the next three years there was no sign of pus or trouble or anything of the kind. The young man went to the College of Physicians and Surgeons, New York, to study medicine, where early in April, 1904, having had no trouble whatever for three years, he was taken with an attack of grip while on ambulance service; had pain and signs of pressure over the right eye in the region of the scar of the former operation. He was put in St. Luke's Hospital, New York. Incision made. Some pus found. This incision was

allowed to close, then reopened, and fistulous opening had been maintained for three weeks at the time I saw him.

This was on May 20, 1904, at which time examination showed a fistulous tract, very narrow at the soft parts but leading into a wider, but not very large, cavity in the bone, apparently not communicating with the nose, of limited area, and very painful to the touch. Managed to curette a little. Enlarged the opening sufficiently to get a tube 2½ mm. into the opening. Curetting of this area was very painful. It was evident that no healing was going to take place from the bottom until the external wound was enlarged and the drainage improved.

He was put into the hospital and again given ether. Reopened along the incision of the New York operation. Dissected up the periosteum. Found the surface bone soft, the sinus obliterated on the periphery over toward the eye, but at the centre soft and containing punky, purulent material. No fluid pus. Cut away the bone almost the entire length of the sinus outwardly as far as over the supraorbital foramen, but not injuring it. Just about the centre of the forehead found one cell containing a little pus. At first I thought this cell led into the right sinus, but it only led to the median partition, which was a little to the right of the exact centre. Then dissected downward, opening one large cell leading to the nose, which contained more or less soft material. Almost the entire front wall of the sinus was taken off, leaving a little bit overhanging so as to retain the ridge. A large opening was made down into the nose and all ethmoidal cells destroyed, and a large size gauze wick was put in. Apparently sound bone left everywhere. Compared with the preceding operation of three or four years ago the sinus was smaller, showing a portion of it to have filled in with solid bone. The centre had evidently remained open and had been reinfected through a small tract, probably from the nose, which opening had not been obliterated.

The after-history of the case is uneventful. There was some pain and photophobia, but no iritis. Cavity was kept firmly packed with iodoform and balsam of Peru gauze, the external opening being maintained until absolute obliteration took place, which, so far as is known, is the case. On September 10, 1904, there was apparently complete healing. There was no discharge from the nose. The sinus was completely closed in. Twelve months have since elapsed without any further trouble. As in all of the other operations there was repeated breaking out of pus from the external wound, and as there has been none this time since discharged, I feel that the case has been completely healed.

Criticism. This case illustrates very well the necessity of complete obliteration of the sinus, as there had been on previous occasions two apparently complete cures after the two former operations, and after the last no recurrence for three years. As,

however, the centre of the sinus had not been entirely obliterated and there was still some communication with the nose, there was a chance for reinfection, which took place. The mistake which I made in the earlier instances was in allowing the external wound to close, so much so as to be unable to work through it satisfactorily without being satisfied that every bit of the centre was filled with firm bone before so doing.

CASE XIV.—Mrs. B. Patient seen by Dr. S. on January 13, 1905, complaining of pain in the region of the right eye and right frontal. Next day right eye bulged very much forward. Next day bulging still the same. Pain rather diminished. Considerable pus discharge. Seen by Dr. J., an oculist, who thought the condition was probably frontal. Seen by me on January 16th. Examined carefully in bed. Slight frontal sinus tenderness; no pus in the nose; right eye protruding very markedly. Deemed it advisable to open the right sinus as an exploratory measure.

Admitted to the hospital. Temperature normal; pulse 68. Opened sinus in the internal angle under the ridge. On opening found some grumous-looking pus, not especially great in quantity, which welled forward but was apparently not under much pressure. Entire sinus did not extend very much upward, but a probe easily went down in the region of the orbit and behind it. Enlarged the bone incision with cutting forceps so as to take away a portion of the nasal process of the superior maxillary. Made large opening into the nose, which opening was apparently closed, as a probe would not go through it at first. Extended bone incision upward, and removed major portion of the anterior sinus wall, curetted out the entire mucous membrane, which was degenerated. Found that there was a good-sized opening into the opposite sinus at about the root of the nose. Opened the left sinus in a similar manner, making incision through the opening downward about as far and upward toward the notch of the supraorbital nerve, but not as far as this. Good-sized opening made into the nose, large enough to admit very large curettes. Scooped out all the degenerate mucous membrane. Packed both sinuses with gauze.

January 19th. Dressing not done for three days. Temperature has been normal. Practically no pain. On removing the dressings no sign of pus. Eye has gone backward to its natural position to quite a degree. Is not yet quite normal.

22*d*. Changed the dressings. Not much attempt at healing as yet.

27*th*. Some pus discharge on the dressings, which have a little odor at the end of two days. Right eye has resumed nearly its natural position. The only swelling seems to lie in the partition between the two sinuses curetted. There is free drainage into the nose. Patient has normal temperature; is feeling well.

28*th*. Is beginning to form granulations on the floor of the sinus all around. Sinuses do not extend upward a great distance. Cut

away small amount of bone with the Kerrison forceps. Patient's general condition all right.

March 31st. Considerable degree of filling in has occurred. Sinuses now very much smaller. Do not extend outwardly more than half an inch from the external opening. Right one extends upward a short distance. Left one practically filled except for the centre. Eye apparently normal.

June 24th. Left sinus entirely filled, right one open about the size of a large pea, still communicates with the nose. Now filling rapidly. Patient has been at work for several weeks. Scar on the left side inconsiderable.

July 15th. Complete healing; only slight scar.

While the treatment in this case lasted a considerable length of time, from the middle of January until the last of June, which length of time might be considered as a fair criticism of the method, it must be remembered on the other hand that each sinus was of large extent and that complete obliteration has been obtained without scarring to any extent.

The case which follows was of peculiar interest to me because it is the only one I have ever seen in which the empyema of the frontal sinus produced an external fistula. It also shows that a considerable degree of relief and comfort and, from the patient's standpoint, almost a cure can be brought about by merely improving the drainage.

CASE XV.—W. F. T., aged sixty years. Came November 26, 1904, on account of a sore in the external nose about midway between the bridge and the angle of the eye on the right side, which was discharging pus. This had lasted for several months. He had been treated by two ophthalmologists for abscess of the tear duct. Examination with the probe showed this space to have no relation whatever to the tear duct but to lead upward into the frontal sinus and downward into the nose. Was able to syringe directly from without into each cavity. Examination of the nose showed the whole condition to have been due to a polyp or bullous-like formation of the anterior end of the right middle turbinate, obstructing the natural outlet from the sinus. The sinus was washed out several times through a cannula. The anterior end of the right middle turbinate was then removed, and as the symptoms were disappearing he did not wish any radical operation done.

December 3d. Rewashing of the sinus brought away dark mucopus, but the fluid pus seemed to have disappeared for the time being, and the external wound had healed, although the probe easily traversed the region around the sinus. There was more or less bare bone around the opening from the external nose. Radical operation was urged but he did not feel as though he could afford to lie up for permanent operation.

January 31, 1905. The external wound seems to have permanently healed. The discharge now comes entirely down through the nose and he is having no conscious trouble with it. The case is by no means cured, but as it is not troubling him any he declines to have anything more done.

Careful examination of the case showed that he had presumptively had a chronic sinus trouble for a good many years, the pus discharge being denominated as merely catarrh, and up to the point where the internal drainage was interfered with and the pus broke through externally, he was not conscious of much if any trouble. This case illustrates the fact that many persons will stand a pus discharge from some one of the accessory sinuses without being conscious of very much trouble for many years, and only seek relief when some concurrent inflammatory condition blocks the outlet and causes absorption of the pus. In this instance the patient thinks that my operation of the removal of the middle turbinate has cured him, whereas, as a matter of fact, I have only made the drainage from the cavity better.

Of the cases cited those operated upon, namely, Cases V. to XIV., inclusive, briefly analyzed show the following results: In 7 the sinus was opened at the inner angle of the eye, nasal drainage established, with subsequent closure of the external wound after a varying period, the so-called Ogston-Luc method or some modification of it. Of these 2 showed a cure without recurrence, 4 had one or more recurrences with final cure, 1 certainly by obliteration, the others probably; 1 is still under treatment but is practically cured, but has had more or less recurrence. Three cases were treated by the method of obliteration, and of these there have been no recurrences. The cases which showed recurrence were all of them accompanied by more or less ethmoidal suppuration or occurred in connection with maxillary antrum suppuration. In the paper by Logan Turner already referred to he states that in 62 per cent. of 172 cases, histories of which were examined by him, the ethmoidal cells were implicated, and that in 55 cases operated upon by the Ogston-Luc method there were 33 cures and 22 failures, showing probably that the successful cases were those in which there was little or no involvement of the ethmoidal cells, while the cured cases were those of pure frontal sinus trouble without ethmoidal involvement.

My own statistics are of course too few of themselves to generalize from. In the main, however, it would seem to me that the cases in which the simple opening and drainage of the sinus have been immediately successful were those in which the ethmoids were involved either but little or not at all. The cosmetic results have been good in all the cases, even those where there were several recurrences.

Conclusions. What then shall be our attitude with reference to the treatment of chronic frontal sinus suppuration? What operation would seem to offer the best chance for the patient? Certain things must enter into the consideration of each individual case, such as the length of time the suppuration has lasted, the degree of discomfort, the probable danger to life, and the age, sex, and social condition of the patient. The intranasal method of treatment should certainly be tried if it offers any chance of a permanent cure or of sufficient degree of relief to satisfy the needs of the patient. If persisted in for a reasonable length of time without result the question of external operation should be considered.

Of the external operations, viewed in the light of the past and present experiences of the majority of rhinologists, what is the one to be chosen? In simple suppuration, not long continued, I agree with the conclusion of Luc as referred to by Logan Turner, that in cases where the dimensions of the sinus are limited and the frontonasal canal is roomy and the ethmoidal labyrinth free from suppuration, and where the inflammation has not lasted any considerable length of time, the simple operation of opening and draining the sinus, with closure of the external wound, will effect a satisfactory cure. In all other cases, especially those associated with ethmoidal trouble, accompanied by the formation of polypi and the probable implication of the sphenoidal or maxillary sinuses or both, it seems to me, in the light of my own and the experience of others, that some form of the obliteration method is the operation to be chosen. Whether it shall be that which I have myself performed, namely, the taking away of a sufficient portion of the anterior wall of the sinus below the supraorbital ridge to gain access to the sinus, and so much of the floor of the nose and the nasal process of the superior maxilla as may seem necessary, followed by a prolonged packing, a method which, while reasonably certain to effect a cure, necessitates a disagreeable and long-continued amount of after-treatment if the sinus be at all large, especially long and disagreeable if both sinuses are affected, or the method of obliteration described and followed by Dr. Coakley, namely, the taking away of the anterior wall of the sinus above the supraorbital ridge with immediate obliteration and closure of the frontonasal duct, partial suture of the skin opening, with packing of the cavity until obliteration takes place, this being materially aided by the partial sinking in of the soft parts, or the more radical and much discussed operation of Killian, must be determined by the individual case, the social status of the patient, and the individual skill and proclivities of the operator. The decision in many cases can hardly be made prior to the time the patient is on the table and the sinus opened, as, although transillumination gives us considerable information in many cases, it frequently does not absolutely show the extent of the sinus, hence the operator should

be sufficiently conversant with all the methods of treatment so that he may at the time of the operation be prepared to do that one which, in the light of his knowledge, then and there offers the best results for his patient. The question of the cosmetic result must be carefully considered, as cosmetic results which would be considered good in Germany would not be so considered here.

The published statistics of Killian's operation seem to show it to be satisfactory in its results, at the same time not all of the cases showed immediate cure, and in some of them there was more or less pus discharge for a considerable length of time thereafter. Killian's operation is also not wholly devoid of danger, as Freudenthal has recorded cases where he found the dura instead of the sinus in opening up above the bridge after the method of Killian. This of course could be obviated by invariably opening against the inner angle of the eye, where, if any sinus is present at all, it will be found. Of the cases reported as cures I think some of them may justly be doubted whether they are cures or not, as so long as the discharge from the nose has not entirely stopped the case can hardly be considered as an absolute cure, even though the patient feels no discomfort. This may be illustrated in the last case reported by myself where the patient regards himself as cured, although that is far from being the case. Better drainage does not necessarily mean a cure, although it means relief.

Another operation has recently been brought to my attention by Lothrop. This is a modification of the Ogston-Luc operation, which seems to have all the advantages of the latter without the danger of recurrence of symptoms of obstruction. At the same time it possesses the advantages of the Killian, but is not followed by the deformity which this operation produces. A short incision is made under the inner end of the eyebrow so as to approach the orbital floor of the frontal sinus. The sinus is opened by this route and the whole of the nasal floor of the frontal sinus is removed chiefly by means of curettes, thereby taking away all the ethmoid cells situated in the vicinity of the ostium frontale, limited externally by the inner wall of the orbital fossa and internally by the inner wall of the lateral mass of the ethmoid, a part of which is removed. This opening embraces, therefore, as great a space as the anatomy of the parts will allow. The anterior wall of the sinus remains intact. Lothrop states that none of these operations have been followed by obstruction and that drainage is free.

It is also fair in considering any report of cases to assume that in many patients it may be impossible to get a perfect cure, as the anatomical conditions and pathological changes militate against this and do not allow the operator with safety to his patient to remove a sufficient amount of tissue. I think the operator should feel fairly well satisfied with his results when he has got his patient free from recurrence, free from annoying nasal dis-

charge, free from pain, even though there may still continue to be a moderate amount of ethmoidal mucopus which, in the majority of instances, will not be noticed by the patient as anything more than ordinary nasal discharge.

The whole subject of nasal suppuration demands more consideration from the general practitioner than it has yet had, and in view of the fact that nearly every winter epidemics of grip bring about attacks of acute sinusitis, many of which can be prevented from becoming chronic under proper methods of treatment, it becomes the duty of members of societies such as this, by their written and spoken word, to educate the average practitioner into the need of proper nasal treatment for these conditions in order that they may be recognized early and cases of acute sinus inflammation prevented from becoming chronic.

THE LARYNX IN TYPHOID FEVER.[1]

By Chevalier Jackson, M.D.,
of Pittsburg, Pa.

The vital importance of this subject is better stated by and comes with better grace from a practitioner of internal medicine. Dr. John W. Boyce wrote to the writer as follows:

"The question of great interest is whether it is possible for these affections to cause a fatal termination without such symptoms as shall plainly indicate the nature of the occurrence.

"Theoretically, it seems impossible that a man should choke to death without the conspicuous symptoms of asphyxia, particularly cyanosis; but observation has convinced me that it is only too true; that these cases may sink away as peacefully as a baby goes to sleep, and that one may not in the least suspect what is happening. I am convinced that in times past such has been the case, where I attributed the death to collapse. In the first case of this nature that I ever recognized (though it is scarcely possible it was the first case I ever saw) a fairly acute resident physician had seen the patient, and had no idea that his dyspnœa was in any way an important matter. The case was taken to the operating room for tracheotomy as a precautionary measure, not with the idea that the operation was needed but for fear it might come to be during the night in the absence of a physician. While we were making leisurely preparations for a systematic operation I accidentally noticed that the patient had stopped breathing. Fortunately the surgeon was

[1] Read at the meeting of the American Laryngological, Rhinological, and Otological Society, Boston, June, 1905.

standing there with knife in hand and was able to stab the patient on the instant. There would not have been time to send to another room for an instrument; as it was, artificial respiration was performed for fully ten minutes after the trachea was opened before the patient could be trusted to do his own breathing. In the last case I saw the patient was rapidly sinking into apnœa, which certainly would soon have been fatal had we not been watching him especially on this identical point; and we had been put on our guard by a slight change of voice, not to hoarseness, but to an aphonia, which I have been accustomed to attribute to relaxation of the vocal cords from extreme weakness and not to lesion. An interesting feature in this case was that the blood pressure as determined by the sphygmomanometer rose when the trachea was opened instead of falling, as we might expect when asphyxia is relieved. Yet there is no doubt whatever that this patient was in desperate need of air before the operation."

LITERATURE. Instead of a review of the literature, which space forbids, a bibliography is appended. The statements below differ from those in the literature radically in many points. The writer can only say that the following is a record of clinical observations as carefully made and recorded as limited time would permit.

The great prevalence of typhoid fever of the severest type in Pittsburg at all times afforded good opportunities for study. The following observations were made in the wards of the Western Pennsylvania Hospital.

In weighing these observations it must be remembered that they were all made on hospital cases which on admission are characterized by foul oral sepsis, severe toxæmia, and an aggravated type of disease due to neglect, overexertion during the early stages, unregulated diet, unhygienic surroundings, etc.

Three hundred and sixty cases were watched throughout their illness. Three separate types were observed. Subacute laryngitis, without solution of continuity of the mucosa; ulcerative laryngitis; perichondritis with or without chondrial necrosis.

LARYNGITIS. Subacute catarrhal inflammation of the laryngeal mucosa was present in 227 (63 per cent.) of the 360 cases. It seemed similar to that occurring as an independent affection, save that the surface was at all times freely bathed with secretion, much of which came from the deeper air passages.

That the larynx should participate in the general catarrhal state of the various mucous surfaces, especially the respiratory surfaces, seems natural. Were it not that a few cases develop more serious and possibly fatal lesions the subject might not merit the two years' study here recorded.

ULCERATIVE LARYNGITIS. *Frequency.* The frequency in private practice is unobtainable, but it must be much less frequent than in hospital experience. No reliable records of observations, system-

atically made on a large scale, are to be found in the literature. The frequency might be supposed to vary in different epidemics, different countries, and in different conditions and environment.

Number examined	360	cases.
Ulcerative laryngitis in	68	" (18.9 %)
Started as abscess of larynx in	4	" (5.9 ")
Required tracheotomy	8	" (11.8 ")
Perichondritis with necrosis in	6	" (8.8 ")
" without necrosis in	11	" (16.2 ")
Abscess from perforation in	2	" (3.0 ")
Ulcerative trachitis in	9	" (13.2 ")
" " with perforation and emphysema in	1	" (1.5 ")
" " with abscess thyroid gland	1	" (1.5 ")
Pus foci in remote locations in	15	" (22.0 ")
Associated with acute purulent otitis media	11	" (16.2 ")
" with leukophlegmasia	6	" (8.8 ")
Died	4	" (5.9 ")

BACTERIOLOGY. The typhoid fever bacillus, unaided by other organisms, has been proven to be pyogenic by a number of observers, though it probably is not often active as such without the aid or subsequent activity of pyogenic cocci. It has been found by various observers in abscesses in almost all tissues of the body, and by these observers is regarded as a prime factor. To the writer it seems reasonable to suppose that, excluding one infection (syphilis), the conditions of the soil are more active factors than the seed in any infection. In typhoid fever we have the conditions of soil for the rapid development of pus cocci. In the cases examined the bacillus typhosus was found in the saliva and in the mucosa, but not in the submucosal abscesses. It is not argued from this that it could not have been the prime factor next to conditions of soil. It is more likely, however, that the ever-present pyogenic cocci found a foothold in an abrasion of the mucosa or in an inflammation of the mucosa due to typhoid bacilli lodged from the blood flowing through the mucosal vessels; or a low resistance of cells or juices due to toxæmia; or to a necrosis of the mucosa from anæmia due to embolism or thrombosis, or to an anæmia from any other cause. Or the typhoid bacillus by causing a non-inflammatory tissue necrosis may have prepared the soil for other more active pyogenic bacteria.

The bacillus typhi abdominalis was found in the throat, both on and off the lesions; in secretions and mucosa. It was not found in any greater numbers, however, in cases with throat lesions than in those without. Pus cocci were, however, much more numerous in the cases with throat lesions, after, not before, the ulcerations developed. Of the 4 cases of abscess 2 were found pure streptococcic, 1 pure staphylococcic, and 1 these two mixed.

In one case tracheotomized for stenosis, a subglottic abscess communicating with the necrotic first tracheal ring was found. Pus withdrawn from this abscess with a hypodermic syringe before evacuation proved the pus to contain a pure culture of streptococci. Careful search showed no Eberth's bacilli.

Of the 11 cases of ulceration that also had acute suppurative otitis media it is a striking fact that in every case the same pyogenic organisms were found to preponderate in the laryngeal lesions. Thus of the 2 abscess cases that also had ear suppurations streptococci in pure culture were found in both. In the other 9 the laryngeal ulcerations, being superficial, were necessarily mixed infections, but the preponderating organisms corresponded to those of the ear. Sloughs examined microscopically in 6 cases swarmed with staphylococci, in 4 other cases with streptococci, neither being in pure culture, but mixed with other cocci. Klebs-Loeffler bacilli were found in 1 case only in this gray slough, and were not found in one case where there was an exudation in the trachea.

The following pathogenic organisms were found in the sloughs from the laryngeal ulcers: streptococcus, staphylococcus aureus, staphylococcus albus, pneumococcus, and bacillus diphtheriæ.

Laryngoscopic Appearances and Clinical Pathology. Anyone who has watched these cases can make a diagnosis of typhoid fever by the laryngeal appearance and the odor of the breath, which is peculiar, rather offensive, but not putrid. It is as distinctive as that of diphtheria.

Laryngoscopy is not difficult to one accustomed to work, as every laryngologist should be, with the patient in the recumbent position. A self-illuminating laryngoscope is convenient. The secretions should be expectorated and the throat cleared by the patient if possible. The tongue must be handled gently, and care taken that its epithelium does not come away with the towel. There are very few cases in which a good view of the larynx cannot be obtained. Œdema and nothing else may be the picture, the ulcers being hidden.

In one case three blebs were noted, one on the posterior surface of the epiglottis, another on the left arytenoid, and one in the posterior commissure. They contained sanguinolent serum and were followed by ulcers which differed in no way from the ulcers originating as sloughs.

In one case a pseudomembranous exudate lined the larynx and trachea as far as could be seen. This could be wiped away and did not leave a bleeding surface.

Four cases of purpuric spots on the laryngeal mucosa were observed. None of these were followed by ulceration. One of them had submucosal hemorrhage, and all had purpuric spots in the mucosa and skin.

Trachitis without ulceration was noted in 126 cases, though many of these were exceedingly mild. In 2 cases the trachea during life was seen to be ulcerated. One of these ulcers perforated, producing emphysema. In 2 cases tracheal ulcers were seen with the bronchoscope after death.

Of 68 cases of laryngeal ulceration the sites were as given below, a few cases having as many as four ulcers, and some cases having large ulcerations covering more than one location.

Epiglottis	42	Anterior commissure	4
Aryepiglottidean folds	22	Infraglottic region	3
Interarytenoid space	18	Trachea	4
Arytenoids	10	Bifurcation	2
Ventricular bands	7	Bronchi	2
Ventricle	5	Vocal bands	1

It is remarkable that the frequency of involvement should be so nearly in direct relation to the distance from the mouth with the notable exception of the vocal bands, which seemed quite immune. The one case in which they were ulcerated had a specific history and cleared up promptly under specific treatment.

As previously stated the larynx of practically every typhoid fever case was the seat of a catarrhal inflammation, and this did not differ from the acute laryngitis with which we are all familiar. In 5 cases this process seemed to become greatly intensified and the mucosa became diffusely crimson in color, so great was the engorgement. Minute vessels ruptured, apparently from coughing. These hemorrhagic spots later became the site of ulcers in a few cases. In many cases ulceration apparently was not preceded by any diffused increase in the intensity of the laryngitis.

Of the 68 ulcerative cases, 18 had ulcerations of the linear type, 6 were Y-shaped or branched, and 5 were wedge-shaped. Some of these cases also had ulcers of the usual rounded or oval outline.

In ten instances the development of the ulcers was watched from their incipiency. A swelling appeared over an area of mucosa, usually elongated, sometimes linear in shape, and from 2 mm. to 15 mm. in length. At first the swollen area was pale, followed in a few hours by redness. At the next stage there appeared a thin grayish slough, as if the mucosa had been lightly brushed with a solution of argentic nitrate. Through this the deeper structure could be seen to be of a dark purplish color. This faded within a day to an even opaque, dirty gray slough, which separated in a period varying from one to three days, leaving a shallow ulcer, with at first illy defined edges, and a dirty gray bed dotted in a few instances and for a short time with dark-red spots. Later the edges became thickened and the bed covered with a dirty gray exudate. In some instances the ulcer became surrounded by an areola of engorged mucosa, but more often the tissues seemed normal close up to the edge of the ulcer. The depth of the ulcer seemed to be indicated by the thickening of the edges. Soon after the ulceration reached the perichondrium œdema set in. The last two statements are based upon examination from below after tracheotomy. In 5 of the 8 tracheotomized cases the ulcers had thickened edges, and the ulceration had reached the perichondrium. The other 3 tracheotomized cases were abscesses, not ulcers.

In 1 case an abscess of the thyroid gland was opened at the tracheotomy, and the first and second rings of the trachea were found necrosed. The abscess was walled off from the neck tissues but was burrowing under the tracheal mucosa, which was pushed inward, occluding the tracheal lumen almost completely. The patient recovered promptly.

ETIOLOGY. In considering the etiology, first place must be given to the typhoid toxæmia. The blood, tissue, and juice changes are of the most profound character, and this must be borne in mind in considering every other element, as it renders operative otherwise insignificant factors. Slight local trauma, or pathologic change, that in health would be promptly cured by nature, becomes formidable in the absence of resisting power.

It is an accepted fact that a particular type of complications may characterize one epidemic of typhoid fever and a different type another. In Pittsburg we have typhoid fever with us all the time and not only as an epidemic. Yet the tendency one season to preponderance of one type of complications is very noticeable. This has to be considered in weighing etiologic factors. There must be a cause for the preponderance. When the pervading type of complication is purulent and occurs in one ward or institution the natural conclusion is that it is infective and that the source is a previous case. This was particularly noticeable in the purulent complications in the Western Pennsylvania Hospital, which nearly ceased after the elimination of flaws in the technique of disinfection of wards, beds, mattresses, pillows, etc., discovered and righted by Dr. Thomas Arbuthnot. There are several weak points in any argument based on this observation. The same tendency to type is noticed in widely scattered cases in different environments. It is well to note the tendency to laryngeal implication in certain diphtheria epidemics.

One case in 68 (1.47 per cent.) occurred before the twenty-first day. Thus, 98.53 per cent. occurred after the twenty-first day. In 8 cases that died before the twenty-first day no laryngeal lesions were found. In 8 cases that died after the twenty-first day ulcerations existed in the larynges of 2. These 16 examinations were made on the cadaver with a self-illuminating laryngoscope on cases in which autopsy was not permitted, and are not included in the 360 cases systematically examined.

These facts point to the existence of an etiologic factor after the third week that does not exist prior to that time.

What are the characteristics of the third week and the following weeks of typhoid fever as compared with the earlier period? Sensitiveness to cold, a rapid, often sudden emaciation, lowest resistance of the tissues and juices, thrombotic phenomena, glandular disturbance, pneumonia, and bronchopneumonia beginning inflammatory and purulent troubles, separation of the sloughs from

the enteric lesions. The worst of the high temperature is over, but its damages remain.

The etiologic possibilities may be classed as follows:

PREDISPOSING FACTORS. 1. Dorsal decubitus.

2. Lessened cough reflex.

3. Lessened frequency of deglutition.

4. Lowered resistance and weakened phagocytosis or its equivalent, due to toxæmia, etc.

5. Increased virulence of organisms proliferating in the air passages.

6. Intensity of the general infection: (*a*) pyrexia and (*b*) toxæmia.

7. Poor blood supply to larynx, predisposing to necrosis.

8. Pretyphoid disease: (*a*) local and (*b*) general (syphilis).

9. Draughts of air, especially when patient is perspiring.

10. Hygienically bad surroundings. Crowded wards.

11. Hydrotherapy: (*a*) sponging, (*b*) tubbing, (*c*) wet-pack, (*d*) ice-pack, and (*e*) ice-bag on head.

12. Drugs.

13. Age.

14. Sex.

15. Nationality.

16. Occupation.

EXCITING FACTORS. 17. Infection *via* (*a*) blood. Typhoid fever bacilli or pus cocci in infected emboli or floating free; (*b*) abrasions from coughing trauma or screatus.

18. Thrombosis: (*a*) aseptic clot (stasis), (*b*) infective emboli (classed above), (*c*) endophlebitis.

19. Oral sepsis.

1. That dorsal decubitus can favor thrombosis by hypostasis the writer does not believe. Anyone who will look along a ward will see that the larynx of nearly every patient is higher than any other portion of his body except his face, varying slightly accordingly as pillows may be arranged. But it seems probable that the sagging laryngeal cartilages may, so to speak, pinch the mucosa between themselves and the vertebra in the supine patient.

2. Lessened cough reflex by favoring incubation of organisms and the development of more virulent cultures in the accumulated secretions is a factor.

3. Lessened deglutition may be a factor in a similar way.

4. Lowered resistance.

The lessened resistance of the tissues in typhoid fever was demonstrated by Brieger and Ehrlich. Malignant œdema followed the injection of a solution of musk in typhoid fever patients, but was harmless in others.

Liebermeister years ago wrote: "If one studies closely the manifold complications and sequelæ of typhoid fever, in the living and at autopsies, he will receive the impression that in severe cases

the resistance is reduced to the minimum in all organs without exception, and that there is an extraordinary tendency to the destruction of the tissue." This expresses the clinical aspect of the predisposing factors. It is a condition probably brought about by the action of the toxins on the tissue cells, and is evidenced by the rapid formation (often in twenty-four hours) of large abscesses in various parts of the body. That the vulnerability of the respiratory mucosa should be similarly increased is a reasonable conclusion, while it is, more than other tissue, exposed to pyogenic cocci.

During the time these observations were in progress careful watch failed to find laryngeal ulceration in any other than typhoid fever cases with one notable exception, a case of pulmonary tuberculosis, which was sent in through error and could not be got out. Laryngeal ulceration developed in this larynx while in a room just off the typhoid fever ward. The ulcers were not tuberculous and could not be distinguished from those occurring in typhoid fever, except that the typhoid fever breath was lacking.

5. The possibility of increased virulence was not investigated, but it is deserving of research.

6. As stated, the chief etiologic factor is the typhoid fever. This renders the mucosa vulnerable and thus permits activity of other factors which otherwise would fail. Unlike the complications of the exanthemata, ulcerative laryngitis complicating typhoid fever bears as to the likelihood of its occurrence, its course, and its termination, a close relation to the severity of the primary disease. Under the head of frequency it was mentioned that inquiry among physicians in general practice developed the fact that those practitioners in the poorer districts occasionally saw cases of laryngeal complications, while many of those practising among the wealthy class never saw even one case. The reason for severe cases in the poorer districts seems to be the continuance at labor in the earlier stages (some men working with a temperature of 103°), and at the same time eating a general diet, often forcing food down in spite of anorexia, in the ignorant hope of keeping up the strength. Many laborers make an untreated walking case out of the early stage conditions that would put the wealthier man in bed with bi-daily visits of the physician and the constant attention of two trained nurses to enforce dietary and other restrictions and a proper regime. Another reason may be in the filthy habits and unhygienic surroundings of the poor. The importance of the bathtub in prophylaxis is unquestioned.

The writer's records show that out of 68 cases of ulcerative laryngitis 61 (89.7 per cent.) occurred in cases in which the temperature exceeded 104.2° every day for a week or more, and in 23 (33.8 per cent.) the temperature reached 105°. All other symptoms in a fair proportion of the cases kept pace with the temperature, as indications of the severity of the case. No case of laryngitis occurred

in any case in which the temperature did not exceed 103°. In considering the temperature it must be remembered that except in a few cases not thought to require it the patient was sponged every time the temperature rose above 103°. Wet-packs, and in some cases ice-packs, were used if sponging failed to bring the temperature down.

Von Ziemssen thought the pyrexia was the most important factor in lowering resistance of tissues and advocated antipyretic treatment as the best mode of preventing laryngeal complications. As to the causative influence of pyrexia, this was disproven by the writer's records. Of 10 cases allowed (for other reasons) to go without sponging there were 2 (20 per cent.) developed laryngeal ulcers. This is about the same as the record of all cases, 68 out of 360 (18.8 per cent.). So that it would seem that it is the toxæmia that lowers the resisting power, and that the pyrexia is but an index of the toxæmia.

As to the effect of antipyretics other than hydrotherapy no records were obtainable, as none other is used by the staff at this hospital.

7. The etiologic possibilities of poor blood supply must be considered in the light of pathologic stasis.

8. Local diseases of the upper air passages were faithfully recorded but seemed to have no bearing except on three points. Of 68 ulcerative laryngeal cases, 10 (14.7 per cent.) were mouth breathers, 13 (19.1 per cent.) had chronic laryngitis, and 66 (97 per cent.) had a foul oral sepsis, with foully carious teeth; and of the 6 cases of perichondritis, 3 (50 per cent.) were hucksters, with a chronic laryngitis when admitted.

Several observations seem to point to syphilis being a factor in the etiology of ulceration. Histories, proverbially unreliable, were of even less than usual aid, because of toxæmic stupor, lack of an interpreter, which often rendered it impossible to separate syphilis and gonorrhœa.

Giving the record, however, for what it may be worth, specific history was: positive in 8 cases (11.7 per cent.), negative in 41 cases (60.3 per cent.), unobtainable in 19 cases (27.9 per cent.).

Specific treatment was of prompt and decided benefit in nearly all cases. But mercuric preparations and the iodides are good excretory stimulants and would render good aid in whipping up the overworked excretory apparatus and thus help to rid the system of typhoid fever toxins. This weakens the test value of therapeutics. In general syphilis favors suppurative conditions; so does typhoid fever. The concurrence of the two probably intensifies the tendency.

9, 10, 11, and 12. A chart of the beds showed that one bed located close to a door was occupied by seven successive cases of ulcerative laryngitis. No other one bed had more than two to its credit. This

door opened on to a short, draughty corridor which led to a room filled with scrubbing sinks, sewer connections, and water-closets. This corridor, on account of defective plumbing (since refitted), was always overventilated, which caused a draught to strike the bed in question. The proportionate share of the defective plumbing, the draughts of air on the sweating patient, and the defective sterilization of bedding which existed there the writer is unable to state, for all have since been corrected; and while acknowledging a weakness for experimental research, he could not, of course, endorse the reinstatement of these horrors one at a time to determine the share of each. He prefers to put them all down as etiologic factors of the first importance.

Overcrowding of wards is certainly an etiologic factor.

In regard to the effect of a draught of air he must acknowledge his inability to demonstrate wherein a draught of air on a perspiring skin differs from a cold wet-pack. If exposure to draughts of air under certain conditions may cause laryngitis in the well, why may they not so act in the typhoid fever patient?

It is Dr. John W. Boyce's practice to omit sponging in certain cases, no matter what the temperature.

He also does not advise the ice-pack. He stimulates and feeds sparingly. Other members of the staff feed and stimulate more freely, the stimulants being chiefly strychnine and whiskey, and some of them use ice-packs freely. Various drugs such as acetozone and guaiacol were tried extensively by Dr. Lawrence Litchfield. Some patients liked an ice-bag on their heads and were allowed to have it constantly, while others objected to it and it was not used.

Detailed tables would be too voluminous, but the result of observations of all these differences in treatment and management led to the conclusion that hydrotherapy, including ice-packs and head ice-bags (tubbing not being used); drugs, including stimulants, and foods and feeding are absolutely negative as etiologic factors in ulcerative laryngitis, with the single exception of the ice-pack, which had some evidence against it.

13. The age of the patients with ulcerated larynges ranged from sixteen to fifty-five years, but 71 per cent. of them were between the ages of twenty and thirty years.

14. In the female wards 3 cases of ulcerative laryngitis developed in 60 cases of typhoid fever. In the average the typhoid fever cases in the female wards were of a less degree of severity than those in the male wards, probably on account of the earlier taking the bed in the initial stages and other recognized causes. These 3 were very severe cases. As this simply brings us to the severity of the general infection, it cannot be said that sex is a factor.

15. The bearing of nativity on the etiology is interesting. Of 68 cases of ulcerative laryngitis the following were the nativities:

Arabia	1	Russia (Northern)	2
Austro-Hungary	25	Slavonia	...
Ireland	2	Syria	1
Italy	21	United States (negroes)	3
Poland	4	" " (whites)	2
Russia (Southern)	7		

The preponderance of the natives of Southern Europe is interesting, though environment and the fact that they now constitute our laboring class must be considered.

16. *Occupation.* It is worth noting that 3 cases of perichondritis occurred in "hucksters" who call their wares loudly from a wagon in the streets. They had severe chronic laryngitis with hyperplasia and dilated mucosal capillaries on admission.

17. *Infection.* There is much argument in the literature to show that the laryngeal lesions are true typhoid fever glandular involvements similar to the intestinal lesions. Local infection of the lymphoid tissue of the larynx by the bacillus typhi abdominalis is of course a possibility, but if so, in view of the cases here recorded, it must occur late when immunity is being developed.

Of the 68 cases but 1 (1.47 per cent.) occurred in the first week. It was a diffuse inflammation starting in the lymphoid tissue of the larynx. This case was probably a true typhoid fever inflammation of the lymphoid tissue of the larynx, though the local bacteriologic examination was negative. This was, however, the only case that could be so classed. The other 67 cases all occurred so late in the disease that they could not be considered true typhoid fever lesions in the sense that the lesions of Peyer's patches are. Enteric reinfections occur at this late period and the laryngeal lesions might be regarded in this light, though such is not the writer's opinion. The outline too of these ulcers, as well as the locations, negative the assumption in the literature that the lesions are specific typhoid fever ulcerations, starting in glandular tissue. Of 68 cases, 42 (61.8 per cent.) had ulcerations on or near the free border of the epiglottis where adenoid tissue does not exist.

Local pyogenic invasion assisted by trauma seemed to account for some of the cases observed. Two chief causes of trauma are the "pinching" mentioned under dorsal decubitus and cough. Coughing induced by bronchial conditions was present in nearly all severe typhoid fever cases. This cough is, of course, an irritant to the larynx favoring abrasion as well as inflammation, though of course it is highly necessary to rid the air passages of infective secretions. A longer sojourn than in the normally active state of the cough reflex probably favors the development of a virulent culture.

Two very clear cases of local pyogenic invasion occurred. In both a stream of pus from an acute suppurative otitis media was

draining into the laryngopharynx from the Eustachian tube mouth. The ulcers were extrinsic (confined to the epiglottic and posterior cricoid mucosæ), and disappeared promptly after circumcision of the membrana tympani with adequate "wick" drainage through the external auditory meatus.

18. The period and the suddenness of the onset, together with the laryngoscopic appearances as already described, especially the linear wedge-shaped and branched outlines, point to a local thrombosis as one of the chief exciting factors.

As to the etiology of this thrombosis, whether it be primarily an aseptic clot from stasis, or from an endophlebitis, or an endarteritis, or an embolus (aseptic or infective), the writer has only two postmortem findings to offer. Two cases with the linear and branched ulcerations had multiple infarcts in lungs and kidneys. These were the only autopsies. Of course the arteries of the larynx are not "terminal arteries," and thus are not subject to infarct exactly as those of the lungs and kidneys are, but arterial occlusion would by ischemia favor limited necrosis and bacterial invasion.

An embolic etiology would require an original focus in the lungs or left heart or in the periphery proximal to the laryngeal arteries. It is to be regretted that the occurrence of endocardial and other possible lesions bearing on embolic etiology were not recorded.

Several arguments against the thrombotic pathology might be advanced. The number of cases of ulcerative laryngitis associated with leukophlegmasia was small (6). Yet each of these had ulcerations of the linear type. It might be argued that femoral phlebitis and thrombosis is a common complication in private practice, while laryngeal ulceration is rare. And certainly the writer must acknowledge that he has seen but one case of laryngeal ulceration outside of a hospital. But it must be remembered that he has never had an opportunity of systematically observing the throats of typhoid fever cases in private practice. It may be frequent yet, as pointed out by Dr. Boyce, unrecognized, even though it actually be the cause of a fatal termination.

19. Oral sepsis is closely related to infection. Its part seems so self-evident as to require no proof.

Glossitis was present in 2 cases and in 1 case through extension by continuity was the cause of perichondritis, chondrial necrosis, and abscess with subsequent ulceration.

Symptomatology. The writer considers it his duty to state as a preliminary that all the classic symptoms of laryngeal trouble may be absent, or so slight as to escape notice, being masked by the general malady. Categorically the local symptoms that may be noted are: pain and tenderness, stridor, dyspnœa, dysphagia, odynphagia, dysphonia, odynphonia, hoarseness, croupy cough, and external swelling over the thyroid cartilage. Expectoration is masked by bronchial expectoration.

The general symptoms occasionally seen are recrudescence of pyrexia and extreme prostration with symptoms of approaching death by toxæmia and exhaustion. The pyrexia, as in all post-typhoid fever conditions, usually partakes of the type peculiar to the general malady. In a few of the writer's observations the laryngeal complication only reached the stenotic stage so long after the general toxæmia that the dyspnœa was of the usual active type. But in the feeble state of the profoundly toxæmic typhoid fever patient it was not the symptom we see elsewhere. In 4 of the patients saved from asphyxia by tracheotomy there were none of the symptoms we are accustomed to look for—no sitting erect, gasping, stridor, struggling, violent action of the accessory respiratory muscles, cyanosis, etc. In 3 others slight cyanosis was present and some effort was being put forth, while only in a few cases was the patient making a typical fight for air.

Dyspnœa when present may be due to œdema, occlusion of the respiratory lumen by a projecting abscess, occlusion of the glottis by a fragment of necrotic or sloughing tissue, collapse of the cartilaginous box from necrosis, fixation of, or abductor paralysis of, the cords.

Hoarseness is the most frequent symptom; aphonia next.

The usual cough of typhoid fever was present in 50 (5.9 per cent.) of the 68 cases, but in only 36 of the 50 could it be said to be changed in character after the onset of laryngeal lesions. In a few of these it became quite hoarse and croupy.

The onset of symptoms, averaging all cases that had symptoms, was the thirty-first day. The onset of ulceration in all cases averaged the twenty-second day.

Diagnosis. Diagnosis is easy if the larynx be examined regularly. If reliance be placed on a watch of the symptoms not more than 1 case in 10 will be diagnosticated, and, as pointed out by Dr. Boyce, a certain number will die without the diagnosis ever being made, even in some of the instances in which the laryngeal condition is the real cause of death.

Prognosis. As to life, the prognosis is good if considered apart from the primary general infection. If watched by one skilled in the use of the laryngoscope there is little danger if the suggestions given under treatment be followed. Practically, however, it is not possible to separate the prognosis, and when it is considered that the laryngeal lesion appears in practically always the profoundly toxæmic cases the prognosis as to life must be said to rest altogether on the patient's general condition. As a matter of statistics no case of the 68 died of either the laryngeal lesions or the tracheotomy done for their relief. Only one tracheotomized case died and that was of toxæmia. Of the 68 cases, 4 cases (5.8 per cent.) died; 1 of relapse with intestinal perforation, 2 of toxæmia, 1 of pneumonia. The mortality (5.8 per cent.) seems low, considering that all were severe

cases, but it must be remembered that the laryngeal ulceration only appeared after the patient was convalescent and had run the gauntlet of his greatest dangers. How many early fatal typhoid-fever cases would have had laryngeal lesions had they lived of course is beyond human knowledge. In 3 cases ulceration perforated into the soft tissues of the neck, but in no case was there a cellulitis or violent inflammation of the extralaryngeal tissues.

No case need die from the throat complication. The greatest danger to life is from failing to recognize the existence of a laryngeal lesion. Tracheotomy is wrongly regarded as an operation dangerous to life. It has been credited with too many deaths that were justly attributable to the condition calling for it. Of course, as it is the profoundly poisoned cases that get ulcerative laryngeal lesions the prognosis as to life must be guarded, but not because of laryngeal dangers.

As to Voice. In 45 out of the 68 cases (66.2 per cent.) the ulceration healed without alteration in voice or structure, no visible scar remaining. Permanent deformity, without loss of voice and without stenosis, resulted in 8 cases. Stenosis requiring surgical relief to enable the abandonment of the tracheal cannula resulted in 5 cases, all of which recovered good, useful voices of rough quality and uncertain pitch. Cicatricial stenosis can always be avoided if the suggestions under treatment be followed. If it should occur from neglect of early tracheotomy it can be cured with good voice by thyrotomy and prolonged intubation. As to paralysis and fixation, they cease to affect the voice ultimately though their stenotic effects cease only by operative measures.

TREATMENT. In those cases of ulcerative laryngitis that are primary specific typhoid fever lesions, and also those that are of hæmatogenous origin, whether by embolism, thrombosis, or blood-borne pyogenic or specific typhoid fever organisms, prophylaxis is hopeless. But even in these cases the writer does not hesitate to say that if closely watched and judiciously treated no ulcerative case need go on to chondrial necrosis and laryngeal stenotic deformity. These occur in the lately discovered cases and in those cases in which a foul state of oral sepsis, an untreated mucosa, and a long postponed tracheotomy favor a great destruction of tissue.

Prophylaxis then must seek to hold in check the mixed infections that run riot.

The Room. The air should be kept as free from pus organisms as possible, and all cold draughts of air should be avoided, especially if the patient is perspiring. Mattresses and pillows, the entire room in fact, should be sterilized by thorough fumigation. In hospital wards overcrowding must be guarded against.

The Patient. Careful and persistent attention to oral antisepsis (asepsis being impossible) is the first consideration. A dentist's aid should be sought, a daily brushing of the teeth with a soft brush

if the gums be tender, followed by swabbing with a one-half of 1 per cent. carbolic acid solution, with borax, sodium bicarbonate, menthol, alcohol, and camphor-water is the most agreeable and refreshing as well as cleansing agent. The patient should be encouraged to clear his throat frequently. All food and water should be sterile. From a laryngologist's viewpoint the ice-pack should not be used unless deemed necessary to prevent a fatal degree of hyperpyrexia.

The writer does not commend the spraying of the nose except where there is a tendency to accumulation of dried or crusted secretions. Usually the natural posterior drainage of the nose, favored by dorsal decubitus, is sufficient to keep the nose clean.

In all the prophylactic measures one thing must be clearly borne in mind, namely, anything that excites or annoys the patient is a detriment to his general condition, so that we must be very sure that what we do is of unquestionable value. The regular toilet of the mouth is agreeable to the patient and is certainly a good thing from the laryngologist's as well as the attending physician's viewpoint, and need not annoy the patient.

In the luetic cases specific treatment yielded prompt results. In other cases considered able to stand it the same treatment yielded good results, probably by acting as an excretory stimulant assisting in the elimination of toxins.

Inhalation of benzoin from hot water is useful. The room may be steamed with water kept boiling to which tincture benzoin compound is added.

In acute œdematous stenosis adrenalin solution 1: 1000 (or any of the similar preparations) as a spray is useful to gain time and may tide over a danger point, but it may become a danger if it lead to too long a postponement of a tracheotomy.

Intubation is useless in œdema, worse than useless in abductor paralysis. If used for the latter, glottic closure will terminate the case promptly should the tube be coughed up.

Of the 5 cases of cicatricial stenosis 3 have been cured by thyrotomy, followed by intubation. They have good though hoarse voices, the tracheotomy wounds are healed and ample laryngeal lumen has been obtained. The method of treatment was given in a paper by Dr. Ewing W. Day and myself, read before the Middle Section of this Society in 1904. One, the last, is still under treatment, and 1 was cured by prolonged intubation with large special tubes.

If closely watched and tracheotomized early, chondrial necrosis with its attendant laryngeal deformity, stenosis, alteration of voice, can all be prevented. All lesions disappear as if by magic after an early tracheotomy. Inability to abandon the tracheal cannula indicates too long a postponement of tracheotomy.

Direct medication to the interior of the larynx is easily accom-

plished through the tracheotomy wound, though these cases seem to recover promptly without medication after the trachea is opened.

Abscesses may be incised endolaryngeally if desired, but preparations for tracheotomy should be first made, as it may become urgently needed. Personally, the writer would advise tracheotomy with subsequent incision of the abscess through the wound.

With regard to tracheotomy it is practically harmless if Schleich infiltration anæsthesia be used, if it be bloodless, if the patient be in the Trendelenburg position for the operation, and the day after. If tracheotomy be attempted with a general anæsthetic it will usually end up with respiratory cessation and a stabbing operation. Then we have added to the shock of a general anæsthetic the loss of blood, lack of time for hæmostasis. A few days ago Dr. John W. Boyce locally anæsthetized with salt solution a boy of fourteen years who had been terrified by being told (prior to admission) that he would die on the operating table. Yet he made no resistance and acknowledged no pain when the writer did the tracheotomy.

Toxæmia and also carbonic acid narcosis lessen the need for a general anæsthetic in typhoid fever cases, though no case should be allowed to go on to carbonic acid narcosis. After the respiratory centres are profoundly poisoned recovery may be impossible.

The writer here dwells at some length, as he feels strongly on this subject. The astonishing records appear in the literature of three different laryngeal typhoid fever cases which were considered too feeble to withstand a tracheotomy, yet they lived more than two days after it was decided that they had not sufficient vitality to withstand the operation. From their histories they apparently died for want of a tracheotomy.

Perichondritis and Chondritis. Perichondritis and chondritis are so intimately associated that clinically they cannot be separated.

The poor blood supply of laryngeal cartilages, lessened by the feeble circulation induced by toxæmia, and still further diminished by stasis, is undoubtedly one of the most important factors in causing necrosis.

Of the 17 cases of perichondritis, 4 started as abscesses and 13 occurred from deepening of superficial ulcerations.

The cartilages involved in the order of frequency were the arytenoids, the cricoid, the thyroid, and the annular tracheal cartilages. As to the involvement of the cartilages of Santorini and of Wrisberg it was impossible to determine clinically.

The epiglottis, while the site most frequently involved in ulceration, was in no case the seat of chondrial necrosis, probably because of its better blood supply.

The writer wishes he could demonstrate pathologically that there is a purulent diathesis. Every clinician sees and knows a type of patient in which every inflammation runs to pus. Many of these cases of abscess and necrosis seemed to be of this type.

Next to this as a cause of chondrial necrosis the writer believes that lues is a fundamental element, not so much as an active lesion as a predisposing factor.

RESUME. From laryngoscopic observations of 360 typhoid fever cases the writer concludes that:

1. Serious and fatal lesions of the larynx are much more frequent than is realized.

2. Death may occur from laryngeal stenosis without even the existence of a laryngeal lesion being suspected in the absence of laryngoscopy. If pain and hoarseness be depended upon a diagnosis will seldom be made. Pain is often masked by toxæmia. Cyanosis and dyspnœa are rare, apnœa is common.

3. Unlike the complications of the exanthemata, ulcerative laryngitis complicating typhoid fever bears as to the likelihood of its occurrence, its course, and its termination, a close relation to the severity of the primary disease.

4. The severity of the laryngeal lesion is in direct proportion to the toxæmia, pyrexia not being in itself a factor, but only an index of the toxæmia.

5. Thrombosis of laryngeal vessels in the mucosa or deeper is probably the most frequent local initial lesion.

6. Mixed pyogenic infections are the rule. Laryngeal lesions due to the bacillus typhi abdominalis are exceedingly rare.

7. Prognosis as to life is good if considered apart from the general malady. Not only the life but the laryngeal vocal and respiratory functions will be saved if a tracheotomy be done early. Death from laryngeal lesion means a death for want of an early tracheotomy.

8. Prophylaxis consists in good ventilation, without draughts, sterile bedding, oral antisepsis, sterile food, and water.

9. *Treatment.* Potassium iodide, hydrargyrum biniodid, benzoine inhalations, and oral antisepsis are the best remedies. Early tracheotomy under local (Schleich solution) anæsthesia will cure almost every case.

Thanks are due to Dr. Ewing W. Day for counsel, to Dr. John W. Boyce, Dr. Lawrence Litchfield, Dr. Thomas S. Arbuthnot, Dr. J. A. Lichty, Dr. Percival J. Eaton, Dr. Ellen J. Patterson, and Dr. M. C. Cameron for kind help, and to Dr. Ralph Duffy and Dr. Joseph Barach for the pathologic and bacteriologic work.

In conclusion, the writer urges the laryngoscopic watch of the typhoid fever convalescent or rather defervescent.

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ANGIONEUROTIC ŒDEMA INVOLVING THE UPPER RESPIRATORY TRACT.*

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Angioneurotic œdema, as defined by Collins,[1] is a "vasomotor neurosis or an angioneurosis, characterized by the appearance of circumscribed swellings on various portions of the surface of the body and the mucous membranes, by preference the face, throat, and extremities, without apparent cause or premonition, and non-inflammatory in character."

As it affects the skin, the disease was first described by Quincke as "An Acute Circumscribed Œdema," and from his first description the disease was early called "Quincke's disease." Shortly after Strübing[2] described it as affecting the throat, calling it an "angioneurotic œdema of the throat," regarding it as a vasomotor neurosis, developing rapidly, without fever, after exposure to cold or some traumatism.

The disease is frequently described in books on diseases of the skin under the heading of urticaria. Crocker regards it simply as one of its varieties, giant urticaria. Stelwagon treats of it under a heading of its own, recognizing its similarity in many cases to urticaria, but in other cases says it is entirely or sufficiently independent

* Candidate's Thesis, American Laryngological Association, June, 1905.

of it to warrant its description under its own title. Montgomery and Hyde say it should not be confused with erythema multiforme, erythema nosodum, or giant urticaria. Kaposi and Shoemaker refer to it as a distinct disease, but closely allied to giant urticaria, with which, however, it is not to be confounded.

Osler[3] has been greatly interested in the disease, and in 1888 reported a case of angioneurotic œdema, investigating particularly the question of heredity. Since then he has made three notable communications,[4] reporting in the three papers 29 cases, and in the last one reviewing them all under the title of "The Visceral Complications of the Erythema Group of Skin Diseases."[5] In this paper, and based upon his cases, he shows very conclusively that simple erythema, erythema exudativum, erythema nodosum, certain of the purpuras, urticaria, and angioneurotic œdema, are all very intimately related; that the lesions of any or all of these various affections may, and often do, occur in one individual during the course of his disease, or even during one attack of the disease, so that at one time one observer might make a diagnosis of urticaria; in a subsequent attack another may call it angioneurotic œdema, or during another attack peliosis rheumatica. In a word, a patient observed for a number of years is likely to present at one time or another a variety of lesions, all of them, however, vasomotor neuroses, and so he prefers to classify them under the general head of "the erythema group of skin diseases." He divides the complications into two great groups, the angioneurotic and the inflammatory. To the angioneurotic belong the swellings of the fauces, œdema of the glottis, changes in the bronchial mucosa, causing asthma and the colic due to the œdema of the gastrointestinal tract. The inflammatory group are more serious and include endocarditis, pericarditis, pleurisy, pneumonia, and nephritis.

It is unfortunate that so many writers in reporting their cases have used the term "urticaria" in describing "angioneurotic œdema," this being apparent from their description of the lesions and it has led to much confusion.

While all text-books on the skin describe this disease under one heading or another, and nearly all refer to the fact that the lesions affect the tongue, fauces, and larynx, yet text-books on the nose and throat, with few exceptions, have neglected any reference to or description of the disease in any way. This is particularly so in the case of American text-books. Of sixteen of these latter, but three (Kyle, Posey and Wright, and Bosworth) make any mention whatever of the disease, and in but one (Bosworth) is there any description at all.

Case I.—J. B., male, aged twenty-four years, single, blonde, of a marked neurotic temperament, consulted me at 7 o'clock in the morning of April 4, 1893, twelve years ago, because of difficulty in swallowing and speaking, and of a swelling in the throat, which had

come on suddenly three hours before, wakening him from sleep. He had gone to bed feeling perfectly well.

The open mouth, fixed jaw, thick speech, and evident pain and discomfort in speaking at once suggested quinsy.

On examination I found a pale yellow, almost translucent, œdematous swelling of the right side of the fauces, almost completely filling this half of the faucial opening. The parts involved were the anterior and posterior pillars, the right half of the velum palati, the uvula, and the lateral wall of the pharynx of the same side. The tonsil itself was not swollen, though pushed out as in quinsy. Except for the swelling there were none of the signs of inflammation, the color being a pale yellow, the contour alone resembling peritonsillitis. The larynx was normal, except for the slight congestion of the vocal cords; tongue not swollen, but thickly coated; some earache. He complained of a slight dyspnœa, and marked dysphagia. Temperature normal, pulse 90, constipated. Has now and has had some gastric disturbance. Urine 1022, acid, no albumin. He gives this history:

About a month ago, for the first time in his life, there appeared, without apparent cause, a localized œdematous swelling, without pain or redness, just above the olecranon of the right elbow, followed the next day by a similar swelling over the biceps of the same arm. These were soon followed by like swellings in both feet after a long walk, then in both hands; in a day or two another swelling made its appearance in the left shoulder following a bruise. The next swelling was in the fauces somewhat similar to the present one, except that it was on the opposite side and was very much smaller. This, as had those in the skin, disappeared within twenty-four hours of the onset, and he had a respite for three or four days. The next was serious enough to threaten life. At this time there came on rapidly great difficulty in breathing, rapidly increasing, so that he immediately consulted the laryngologist who had been treating him, and who had the previous day applied a zinc chloride solution to the larynx. After examination he was told there was an œdema of the larynx, pale in color, and in appearance resembling a polypus, but which his physician regarded as urticarial. The dyspnœa was so great that it was feared tracheotomy might be required at any moment. However, without any local treatment beyond the use of a cocaine spray and the internal use of salines, the dyspnœa gradually diminished and the swelling disappeared entirely within thirty-six hours.

His tongue and right cheek were next affected. A day or two later, following a bicycle ride, there came on quickly a swelling of the genitals, the scrotum and penis being enormously swollen, as well as the inner aspects of both thighs, extending from the anus behind to Poupart's ligament and over the hypogastrium in front. This swelling was not painful beyond the feeling of distention; did

not burn or itch, and was not reddened. It kept him in bed, and disappeared in forty-eight hours. From this time until I saw him he had several minor œdematous swellings on the hands and feet.

I scarified with a small bistoury the mucous membrane in a number of places, causing immediate relief to the discomfort and distention, and a marked subsidence of the œdema. Ordered discontinuance of cocaine and antipyrine spray, which he was using, and prescribed saline purgatives. Nine hours later the swelling in the throat was almost entirely gone; pulse and temperature normal. Feels well, but there has suddenly appeared since morning an œdema of the upper and lower lids of the right eye, closing it. Two days later there was no swelling whatever in the throat, and none in the eyelids, but one is just beginning over the left mastoid.

The patient, who had come on a visit to his parents from Brooklyn the night before I saw him, now returned, and I did not see him again for several years; but from a letter a few weeks later learned that he had a few slight swellings in hands and feet, but nothing serious. In writing he called my attention to the fact that all three acute attacks in the throat followed the free use of the cocaine and antipyrine spray, which had been prescribed for nasal obstruction, of which he had been having a great deal of late.

These different swellings of the throat and of the skin were all characterized by coming on suddenly; were circumscribed, œdematous, did not pit on pressure, and there was an absence of redness, heat, pain, and throbbing, not at all resembling an urticarial wheal, excepting in the suddenness of occurrence and subsidence.

Family History. On mother's side, grandmother and her sisters had, as children, urticaria severely. One uncle has an attack of it after eating canned fruit. This uncle's son as a child had the disease so badly that "he would go into spasms." Patient's mother, five years ago, had urticaria off and on for six months, being under medical care continually for it. It disappeared after a successful perineal operation. The mother's family have for years regarded urticaria as a family disease. They are also distinguished among themselves by a peculiar gastric affection, which they designate as the "S——— stomach." No other family history of gout or rheumatism.

The attack in which I first saw this patient occurred twelve years ago. He was at that time a director of a gymnasium. He is now a physician, and in reply to a letter written him recently, he writes me, under date of April 3, 1905, that up to about two and a half years ago he had considerable local trouble in the head and throat of a catarrhal nature, but without the occurrence of the previous œdema experienced when he was under my care twelve years ago. On two separate occasions five years ago he had serious attacks of intestinal hemorrhage. These were preceded for a day or more by a feeling of weakness, depression, weight in the epigastrium, followed by a

condition of collapse resembling a moderate degree of shock. This was followed in a day or two by a profuse tar-colored stool, staining the water red, and with it much prostration for a few days, followed by a gradual recovery. For seven years, from 1896 to 1903, he had considerable intestinal trouble, the principal manifestation being the enormous accumulation of gases, which would rise into the stomach and be belched up, very little passing through the bowels. For the past two years he has been gradually improving, very little of the catarrhal trouble in nose and throat, and the gastrointestinal difficulty gradually but steadily lessening.

This patient has had the disease twelve years. He had the typical angioneurotic œdema of the skin, fauces, larynx, and nose twelve years ago, almost requiring tracheotomy on one occasion, and in the nose so badly that for a time the only relief obtained from the nasal obstruction was through the use of cocaine. In addition to the lesions affecting the skin and upper respiratory tract, he had, five years ago and again two years ago, evidences of the same disease affecting locally the gastrointestinal tract, as well as hemorrhages, probably due to purpura, from the intestinal mucous membranes.

His family history shows the influence of heredity in a marked manner. Being a physician, he was greatly interested in the cause of the various attacks, but was never able to determine the immediate cause, excepting that on two occasions he thought the attacks followed traumatism. He never discovered that any particular article of food produced an outbreak.

CASE II.—A. M., aged fifty-five years, a manufacturer; of a decided nervous temperament. He has been under my observation off and on for nasal and throat difficulty for about six years. He came under my care first because of nasal obstruction, which would come on suddenly and as quickly disappear. There was no periodicity noticed about it, and it occurred at all seasons of the year; would last a few hours or days, no sneezing, and none of the usual signs of a cold in the head, nor did it act like hay fever. The peculiarity was that this obstruction came on usually during the night and by morning was gone. He also had suddenly arising swellings in the throat; the uvula and soft palate would become œdematous, and the dyspnœa would waken him from sleep, so that he was often compelled to sit up to breathe. The voice would become thick and husky at times. There was no oppression or rales in the chest. He insisted the dyspnœa was not that of asthma, but due to the swelling in the throat. The condition would usually begin to improve in a few hours, and in twenty-four to thirty-six would usually have disappeared. An examination of the nose showed at times a rather pale œdematous infiltration of the turbinates, sometimes of one, sometimes of all; but on another visit there would be no sign whatever of any thickening, hypertrophy, or œdema. The only con-

stant abnormality appeared to be a thickening of the septum opposite the middle turbinate on the right side. This thickening of the septum and the anterior end of the middle turbinate I removed, and while it improved his nasal breathing, it did not do away with or lessen the frequency of these suddenly occurring obstructions. These nasal and pharyngeal swellings would be preceded, followed, or there would occur at the same time, pale yellow swellings of the skin of various parts of the body, notably the eyelids, the scrotum and penis, the feet, and hands. These attacks would last from a few days to two weeks, and he might be as long as two months, and occasionally has gone six months without an attack. They have been recurring during the past eighteen years. He is at the present time having an attack, and a description of it will answer for all.

It is two months since the previous outbreak. A few evenings ago, while attending a banquet, the left side of the tongue began to swell; on reaching home he took a large dose of alkalithia, hoping to ward off the attack. The following morning he came to my office with the right upper eyelid œdematous and swollen, and the eye closed; it was pale, yellowish, did not burn or itch. No throat or nasal lesions, and the swelling of the tongue gone. That night he awoke, finding the nose obstructed and the throat swollen, and in the morning I saw him. There was a patch of œdema larger than a bean on the left side of the hard palate, and a pale œdematous swelling involving the uvula and the left posterior pillar of the soft palate, extending down the lateral side of the pharynx to the pyriform sinus. The mucous membrane of the left arytenoid was as large as a cherry. The obstruction in the nose had disappeared and there were no œdematous spots here. There was no pain in the throat, but merely a sense of fulness, with slightly obstructed breathing. On the skin there was a swelling under an inguinal truss and the scrotum was swollen. There is, and has been, no disturbance with digestion; bowels regular. Dr. Dutcher examined the urine and reported as follows: "Color pale amber, slightly cloudy, faintly alkaline, specific gravity 1023, no albumin; no sugar; urea, 2 per cent.; slight indican; no excess of uric acid. Microscopic examination showed mucus and an occasional epithelial cell." The œdema in the throat and skin entirely disappeared during the day, and there was no reappearance for three days, when the right upper and lower eyelids became swollen and closed the eye. This is a typical attack, such as he has been having at intervals for the past eighteen years. The swelling in the feet prevents his wearing his shoes and confines him to the house at times. The dyspnœa is often alarming, but he has never had to call a physician during the night. He says the attacks are becoming less frequent than formerly, and attributes this to the fact that he thinks he knows when they are coming on by his urine becoming more acid, and he at once resorts to alkalies. He has watched his diet during all these years, and has finally given

up thinking any specific kind of food provokes the attacks, as he has from time to time excluded almost everything without that making much difference.

Family History. He has two children, both of whom have been under my care. The son had asthma until ten years of age, and I removed the adenoids because of this ten years ago and with an apparent relief of the asthma. I have not seen him since, and his father says he has had no attacks in several years. He has never had urticaria or the angioneurotic œdema as his father.

The daughter had repeated attacks of what I thought was rheumatic pharyngitis, which recurred with great frequency. I was never satisfied with my diagnosis, and as she was under my care before her father, it did not occur to me that the difficulty might be one allied or related to angioneurotic œdema. Her father says she never had hives or his disease.

The patient had one brother, who, during the latter years of his life, suffered repeatedly and severely from attacks of what were diagnosed as "gallstone colic," and had outbreaks of a skin disease, said to have been eczema. Two years ago he fell, injuring one of his knees. One month later, while in apparently good health, after being at stool, he suddenly fell and died immediately. A searching autopsy was made, but failed to find any cause of death. An embolus in the brain was suspected, but was not found, and, as the case came into court through an action to recover from the accident company, the cause of death was thoroughly canvassed. The autopsy revealed no cause whatever from the gallstone colics from which he had suffered. In the light of the angioneurotic disease in my patient, his brother, the history of gallstone colics for years, without a discoverable cause at autopsy, and the fact that, as Osler has reported, suddenly arising œdemas occur in the brain, it is fair to question whether this might not have been such a case.

The next case appears to be a genuine one of urticaria, affecting the pharynx, larynx, and probably the nose, and shows both the similarity and the difference in the lesions and subjective symptoms of angioneurotic œdema and urticaria.

CASE III.—E. R., aged thirty-five years, merchant; of a highly nervous temperament, consulted me June 24, 1904, because of what he called "a sudden swelling of the tonsils and a feeling of impending suffocation at such times." He snored a great deal at times and at others not at all. The day before consulting me he had entertained a number of men at his country house, which had made him very nervous and had drunk quite a little beer, and that night thought he would die before relief could be had because of the sudden swelling of his throat and obstructed breathing. This came on in a moment and gradually subsided, passing off in half an hour. These attacks of dyspnœa and the desire to constantly swallow have been

occurring for about five years. The first attack followed an outbreak of what he called hives all over the body, including the face and lips, which became greatly swollen. Since that first attack there have been no skin lesions: The swellings in the throat had, of late, been coming on every day or two, though at times he would go a couple of weeks, or even a month. He has been treated by a number of physicians without relief. On examination I found a deflected septum, partially obstructing the left nares, tonsils somewhat enlarged, and the uvula slightly elongated. I could discover no œdematous swelling in the throat, nor any other local trouble. He was not having any symptoms at the time of the examination. He had never had asthma or colic. The urine examined the next day was amber, clear, highly acid; specific gravity, 1030; urea, 4 per cent.; no albumin; no sugar; no indican; a very large excess of acid; no casts; no pus.

I made a diagnosis of angioneurotic œdema, prescribed alkalies and large quantities of water, and explained the nature of the trouble, and asked him to come in again on recurrence of the first attack. He did not return, however, and I did not see him again for nine months; in the mean time he had been under the care of several specialists, one of whom partially excised one tonsil, without, however, affecting the sudden swelling of the throat; another proposed uvulotomy, and the third assured him his symptoms were entirely nervous and largely imaginary. When I saw him recently he was having what he called an attack. He came to my office greatly excited; said his throat felt as though it would close up, and he could not breathe. He was greatly alarmed, and I expected to find a great deal of swelling. On examination, however, the tonsils, soft palate, and uvula and larynx were entirely normal, and I was about to conclude that my diagnosis of nearly a year ago was wrong, when I discovered, with the mirror, on the posterior wall of the laryngopharynx three elevations about the size of a split pea. They were raised above the surface, red, not at all œdematous in appearance, but resembled the wheal of urticaria as seen on the skin. Irritating the membrane half a dozen other elevations soon appeared in size from a pinhead to larger than a pea. The patient said there was no pain, no burning, but a sense of the enormous fulness, and he could hardly believe my statement of the size of the swellings. They were evidently urticarial wheals. In him they would be produced by any slight pyschical irritation or annoyance, such, for instance, as being kept waiting at the telephone, or a person with whom he had an appointment being late, or the unexpected meeting of a friend. These attacks would come on immediately, so that he could not speak for a few moments, but would clutch his throat, begin to swallow rapidly, often to the great consternation of the person with whom he was attempting to converse. An attack would begin often with the sense of burning at the end of the penis, with the

desire to urinate, and then this feeling of swelling in the throat would come on.

Family History. His father died a few years ago, and as far back as the patient can remember he suffered from colic, walking the floor nights repeatedly, suffering great pain and the belching of gas because of it. He was said to have had "nervous dyspepsia." He had never had asthma. He died after five days' illness of acute uræmic poisoning without a previous history of nephritis.

A sister of the patient has had urticaria twice during the past two years. She has had a great deal of indigestion with pyrosis.

This appears to be a genuine case of urticaria affecting the pharynx and larynx and the nose. That it may be easily overlooked is seen by the fact that within a year three different specialists who saw him failed to appreciate the condition, probably because no lesions were present when he was examined. Goodale[6] states that urticaria, although observed upon the mucous membrane of the tongue, does not appear to have been found upon the mucous membrane of the nose or throat. Certainly these throat lesions of his resemble the wheal of urticaria observed on the skin. The amount of local irritation and the subjective symptoms produced were out of all proportion to their size, in this respect greatly differing from the lesions of angioneurotic œdema, as seen, for instance, in the throats of Cases I. and II., in which there were ten times the amount of swelling and mechanical obstruction without anything like the amount of subjective symptoms.

The acute circumscribed swelling of angioneurotic œdema, as it appears in the mucous membranes of the respiratory and the gastro-intestinal tract, appears to resemble that on the skin. It comes on suddenly, in a few moments or sometimes minutes, without warning, while the patient is asleep or awake, and the symptoms it produces seem to be largely due to mechanical obstruction. In the nose, the symptoms are those of obstructed breathing, not much sneezing, and no excessive secretion, differing in this respect from hay fever, but resembling it in the suddenness of occurrence and subsidence. Doubtless many cases of this disease affecting the nose are diagnosed erroneously as hay fever, which disease it closely resembles, both in its etiology and pathology. Both are vasomotor neuroses, both are markedly hereditary and occur in persons of neurotic temperament, while the œdema of angioneurotic œdema is usually transient, lasting from a few hours to a day or two; that is not always the case. Bloodgood[7] has reported what appears to be a genuine case of this disease, which he calls "angioneurotic erythema persistens," in which the parts involved in the œdema were the mucous membrane of both maxillary sinuses and the skin of both cheeks. The œdema persisted for months. He opened both antræ through the canine fossæ, finding the mucous membrane greatly œdematous. The relief following this operation was

but temporary, the erythema and œdema recurring until neurectomy of both infraorbital nerves was done. This gave permanent relief, although the same condition appeared later on the skin of the abdomen.

Osler has also reported cases in which the œdema persisted for a long time. Some of these cases of what are sometimes called perennial hay fever may be those of this disease in which the swollen and œdematous mucous membrane of the nose is not transient but persists. Haviland Hall says: "Urticaria is frequently associated with hay fever," and there is certainly a family likeness between hay fever, on the one hand, and urticaria and angioneurotic œdema of the nasal mucous membranes on the other.

The relation of angioneurotic œdema, and, more particularly urticaria, to asthma has been frequently noted. F. A. Packard[8] reported 3 cases of his own in which attacks of asthma alternated with outbreaks of urticaria on the skin, the cough and dyspnœa being relieved upon the occurrence of the hives, and upon a subsidence of the latter a return of the asthma. The asthma was caused apparently by the presence of wheals on the bronchial mucous membrane. Packard made a thorough search of the literature, and, in addition to his own cases, found and abstracted the reports of 34 other cases in which there was a close connection between urticaria and asthma. Throughout his paper he uses, unfortunately, it seems to me, the term "urticaria" to include "angioneurotic œdema."

In a case of asthma under the writer's care twelve years ago, the patient, a man, aged sixty-five years, had had the disease for about thirty years, and had been under the care of many excellent men, but never received more than temporary relief. He was referred to me because of a possible nasal cause, and I found the mucous membrane of all the turbinates hypertrophied, gray in color, at times greatly œdematous, obstructing the nose completely, and at such times the asthmatic dyspnœa was always greatest. There were no polypi and the septum was straight. He was scarcely ever entirely free from dyspnœa during the year I saw him, and at times, both day and night, in his office or on the street, the dyspnœa was so great that he was a most pitiful object. I cauterized his turbinates as well as removed the inferior borders of both lower turbinates, giving him temporary relief only. The asthma soon recurred as badly as ever, and we agreed that the nose was certainly not an important etiological factor. On one occasion he came to the office with one eye closed from an acute circumscribed œdema, and a number of similar œdematous patches on the face and scalp. They were not red, did not pit on pressure, nor did they burn or itch. On inquiry, he said he frequently had these outbreaks, usually on the head, and to which he paid no attention. I failed to appreciate at that time the probable relation of the asthma and the

skin lesions. It is probable that his asthma was due to an angioneurotic œdema of the bronchial mucous membrane, which had become more or less persistent.

As it affects the larynx, angioneurotic œdema becomes immediately a serious affection. It appears to come on during the night while the patient is asleep. Such was the case with two of my patients. It is entirely probable that many obscure and sudden deaths, occurring during the night, are of this nature, and an autopsy, unless this cause was looked for, would fail to find the cause. During the past few years cases are reported by Woodbury,[9] Hallock,[10] Leddermen,[11] Taylor,[12] Chittenden,[13] Ballenger[14] and others, in which a threatening dyspnœa from this cause occurred. Fatal cases are reported by Osler and Griffith.

Griffith's[15] case, in which both father and daughter died of œdema of the larynx from this disease, is reported in the *British Medical Journal*, June 14, 1902, and should be read in its entirety. He reports the case as "Hereditary Localized Œdema."

"The patient, a woman, was first seen by him in 1886, when she was eighteen years of age, because of dyspnœa, hoarseness, and dysphagia, and œdema of the arm and hand. The œdema involved the epiglottis and aryepiglottic folds, obscuring the view of the vocal cords. Tracheotomy was considered, but did not become necessary, and in a few hours the œdema disappeared. She gave a history of having had these transient œdemas, affecting all parts of the body, from birth. There was no periodicity in their occurrence. There was no history or evidence of any other disease, excepting that she was subject to bilious attacks.

"Her father, from his earliest infancy, had the same kind of swellings, and on three occasions they affected his throat: the first attack at the age of twenty-one years, the second a few years later, and the third at twenty-nine years, when he died as a result of it. In this last attack he came home from work, said his throat was swollen, and his physician was sent for. He came, but returned to his office for something to relieve him, but before his return the patient became suddenly cyanotic and died, asphyxiated.

"For sixteen years, from 1886 to 1902, Griffith saw his patient during many attacks affecting different parts of the skin and throat. The swellings were diffuse or circumscribed, usually pale, sometimes pink, did not pit on pressure, and were never bullous or vesicular. Twice the laryngeal dyspnœa was alarming. The tongue and buccal mucous membranes were at times the site of the œdema. The swelling came on suddenly, without warning, and almost as quickly disappeared. Finally, she had an attack, which proved fatal. At 10.30 A.M. she told her mother her throat was swollen, but that it was subsiding. At noon her husband came home, found her sitting on the floor amusing her child by spinning a top, and apparently free from dyspnœa. Suddenly she dropped the top,

tried but could not speak, clutched at her throat, got blue in the face, and died at once. Her mother, who was present at her death, said she died exactly as had her father before her. Griffith saw her a half-hour after death. The features were livid, tongue protruded, some fulness about the neck. The autopsy showed the larynx to be very œdematous, tense, and pale, and the sides of the laryngeal cavity in contact with each other. Transverse sections of the larynx showed that the œdema affected not only the mucous membrane, but the underlying deeper connective tissues and muscles. The œdematous fluid was serous, untinged with the coloring matter of the blood, and the tissues covering the true cords were decidedly affected."

The etiology of this peculiar disease is not at all clear. There are certainly three and perhaps four main etiological factors. Heredity is clearly shown in the classic case of Osler, reported in 1888, in which the disease appeared in five generations, and of the thirty-nine members of this family nineteen had angioneurotic œdema, two dying of œdema of the glottis. Griffith's case, just reported, is a striking one. All of my cases show the element of heredity. The peculiarity transmitted, Osler[5] says, must be either a morbid susceptibility of tissue or an inherited peculiarity of metabolism, or both combined. The individual attacks are probably provoked by some toxin produced within the body, as in the gastrointestinal tract, or in some other organ or tissue of the body, or it may be introduced from without. Gout and rheumatism are often mentioned as causes, but it is very doubtful if there is any connection. It is more probable that this disease is often mistaken for manifestations of gout and rheumatism. A certain poison or a certain kind of food may cause an attack in one person and not in another, or it may not produce a second attack in the same individual. My patients arrived at this conclusion in their own cases. Strawberries, fish, coffee, ice-cream, contact with a jelly fish are all given in the reports of cases as causes, but these cases are almost always simply the reports of single attacks. Exposure to cold and traumatism, worry, anxiety, mental excitement and fright, are doubtless all immediate causes for outbreaks. Antitoxin, as will be referred to later, is probably one of the various causes. These patients are invariably of a marked neurotic temperament. An interesting and an important question is as to the local condition determining, in a predisposed individual, the localization of the lesions, and Osler seems to think this is an important one in any individual case.

Collins[1] found that in 83 cases studied by him, the lesion first appeared in the larynx six times. He says the occurrence on any particular part of the skin or mucous membrane predisposes that part to other attacks.

There has been much question as to whether the colic, which occurs so frequently in the course of the disease, is the cause or the

result of the outbreak as it appears on the skin or in the throat. There is no doubt but that in mostly all cases the colic is the effect of the acute œdema localized in the stomach or intestinal tract. Morris[16] reports a case in which a portion of the gastric mucosa became detached in using the stomach-tube. It was examined histologically and showed an extreme œdema of the interstitial tissue, the lymph spaces and vessels being enormously dilated. The abdomen has been opened for supposed appendicitis when the condition was one of angioneurotic œdema of the intestinal mucous membrane.

The histological examination in Griffith's case of fatal œdema of the larynx showed that the œdema involved not only the mucous membrane, but the deeper connective tissue and underlying muscles. Crocker says that everything points to the disease being primarily a vasomotor disturbance, direct or reflex, central or peripheral; that a spasmodic contraction of the vessels is followed by a paralytic dilatation and stasis or retardation of the circulation in the papillary layer of the skin. Serous exudation then ensues, producing acute œdema. The same thing occurs in the mucous membranes and readily explains the sudden œdema and enormous swelling that comes on in a few seconds or minutes.

This same kind of œdema occurs in the brain and in the kidneys and probably in every organ or tissue of the body. Gruss[17] reports a case in which acute circumscribed œdema caused an acute retrobulbar œdema producing proptosis, and associated with cerebral symptoms. Osler's Case XV. had repeated attacks of aphasia and hemiplegia, the lesions on the skin being those of purpura, urticaria, and angioneurotic œdema during the fourteen years of the disease.

Ten days after the injection of antitoxin, given for prophylactic or therapeutic purposes, eruptions on the skin occur in a considerable percentage of the cases injected. These eruptions belong to the erythema group and are in order of their frequency, urticaria, erythema, purpura, angioneurotic œdema. The joints frequently become swollen, and there are evidences of acute swellings in the mucous membranes of the respiratory and gastrointestinal tracts.

Antitoxin from the same bottle may be injected into several children, and yet but one may have a skin eruption develop later, showing that the serum is not alone at fault, but that there exists in the affected patient a "peculiar susceptibility of tissue" or an inherited tendency toward one of the erythematous diseases; the antitoxin or something in it being simply the poison that provokes the attack in that particular person. Morse[18] reports a case in which, five days after a prophylactic dose of 500 units, the child complained of chilliness, prostration, vomiting, and œdema of the uvula and pharynx, the acute symptoms subsiding in thirty-six hours. Two years before this same child, after a prophylactic dose of 700 units, developed an urticaria of the skin.

It can readily be seen that when diphtheria exists in the throat, an acute œdema developing during the course of the disease would be attributed to the diphtheritic infection rather than to the antitoxin injected a few days before, and yet it might be due entirely to the latter. From our knowledge that angioneurotic œdema and urticaria occur in the throat, there is no reason to suppose that antitoxin causes these lesions in the skin and not in the fauces, pharynx, or larynx. There is every reason to suppose it does, and it is quite presumable that some of the suddenly fatal dyspnœas occurring in diphtheria are the result of the remedy operating on a peculiarly susceptible individual rather than to the disease diphtheria. The real cause of death in such cases would be overlooked, being masked by the disease. It is possible that the local lesions of diphtheria, as well as the toxæmia, might prevent the development in the throat of lesions which the antitoxin might, however, produce on the skin.

The cases which have attracted most attention have been those suddenly occurring deaths immediately following the injection of antitoxin, and usually in persons who did not have diphtheria, but were given prophylactic doses because of their exposure. The cause and effect here are not open to much question, but there has been much discussion as to how and why the antitoxin caused death.

It appears to the writer that in these cases the children have probably an inherited tendency to angioneurotic œdema, that the antitoxin has been the exciting cause of an attack, and that the swellings have occurred in the larynx or the lungs, producing a sudden œdema that causes asphyxiation, or the œdema may have occurred in the medulla or the brain, causing a paralysis of the respiratory or cardiac centres with resulting death.

For instance, Halderman[19] gave a prophylactic dose of antitoxin to a five-year-old child, who did not have diphtheria. It was asleep when the injection was given and almost immediately the lips began to puff up and in five minutes breathing ceased. Ninfong[20] gave a fifteen-year-old boy a prophylactic dose of 1500 units. Ten minutes later the boy became pale, extremities numb, cyanosis developed, the cheeks became swollen, and free vomiting occurred. Nitroglycerin was given and artificial respiration was resorted to, but the boy died thirty-five minutes from the time of injection. Prof. Langerhans'[21] son was given a prophylactic dose of antitoxin, and almost at once he began to vomit and soon died of suffocation. The autopsy revealed the trachea and bronchi filled with stomach contents and the uvula swollen. Adamkiewicz thought death was due to shock or pain resulting from the forcible injection under the skin of a large amount of fluid. Langerhans insisted that death was due to the poisonous action of the serum upon a healthy subject when unneutralized by a pre-existent toxin.

These cases would seem to have had an acute circumscribed œdema; in the first case of the lips and probably of the larynx;

in the second of the cheeks, the stomach, and the larynx; in the third of the uvula, and probably of the stomach; in each case the œdema coming on within a few minutes and causing death in from five to thirty-five minutes. None had diphtheria, all having received prophylactic doses.

Kortright[22] gave a sixteen-year-old girl 10 c.c. Behring's antitoxin, making the injection in the left side of the abdomen. She immediately said she "felt a lump in the stomach," and Kortright assumed she meant that she felt the swelling of the injected serum under the skin; but it is equally probable that what she felt was an acute swelling in the gastrointestinal tract. Five minutes later she complained of tingling and became restless, and immediately had a general convulsion. This was followed by general tonic and clonic convulsions, opisthotonos, cyanosis, and cessation of breathing. Artificial respiration was kept up till heart ceased in three minutes, death occurring in eight minutes from time of injection. An autopsy made eighteen hours later showed the brain and meninges, kidneys, and liver all markedly congested. No foreign body in larynx or trachea.

In Applegate's case, a boy, aged four years, having diphtheria, was given 15 c.c. antitoxin. One hour later he became very restless, vision completely lost, and three hours after injection died in convulsions.

Kortright was of the opinion that his case died because of something undiscoverable connected with the antitoxin or the injection. It is probable that had another child received the same injection nothing untoward would have followed—there being in his case the unfortunate combination of a predisposed patient and an exciting cause. In these 2 cases the localized œdemas were probably in the brain.

If these statements regarding antitoxin be true, it is not a reason or an argument against the use of this specific in the treatment of diphtheria, because it has saved thousands upon thousands of lives which otherwise would have been lost and has probably caused the death of but a very few. It is, however, an argument against the indiscriminate use of antitoxin for prophylactic purposes, because a single death from its administration in a person who has been simply exposed and had not contracted, and might not contract, the disease is frightfully deplorable. When it is used prophylactically, the previous personal and family history of the patient as regards the erythematous skin diseases should be inquired into, and if this develop the presence of or tendency to any of these diseases, then the antitoxin should be withheld until diphtheria actually occurs, or at least until a bacteriological examination of the throat in such an exposed person shows the Klebs-Loeffler bacillus.

Some makes of serum seem to have a larger percentage of skin eruptions follow than others, and we see much less of these erup-

tions now than three or four years ago, showing apparently that the present methods of preparing and "ripening" antitoxin is eliminating some of its deleterious properties, and it may be that in the future antitoxin may become inert as a cause in predisposed individuals, of the production of the erythematous lesions on the skin and mucous membranes.

The treatment of angioneurotic œdema as it affects the upper respiratory tract may be divided into the management of the various acute attacks as they arise, the correction of any local disease which may possibly determine the localization of the lesions or œdemas in the air passages, and the treatment of the constitutional disease back of the attacks. Of the last I have nothing to say beyond the fact that each individual case must be studied by itself, and if possible the underlying cause found and, if it can be, corrected. Osler has called particular attention to the fact that of his 29 cases of the erythema group, 24 per cent. finally died, 5 of the 7 deaths being due to uræmia. He has never seen, however, a case of pure angioneurotic œdema develop nephritis, and the fact is that cases in which angioneurotic œdema is the only lesion throughout the course of the disease are very few. For eighteen years my Case II. has apparently been such a case.

The treatment of the localized œdemas in the fauces, pharynx, and larynx would depend upon the severity. I have used both adrenalin and cocaine locally with apparent benefit, and in more severe cases, scarification of the pharyngeal and laryngeal œdemas. Should the dyspnœa increase and threaten life, tracheotomy should be done without hesitation or delay. Ice locally is probably contraindicated and might add to the œdema. Delbril advises brisk friction of the skin to induce an attack of urticaria on the skin when there is œdema of the larynx. Packard's cases of asthma would bear out the wisdom of this. Quiet in bed, reassurance, and, as far as possible, the removal of anxiety are important. These patients are highly neurotic, and fright alone might precipitate a sudden and fatal termination. Internally saline laxatives and alkalies to reduce the usual hyperacidity of the urine and increase the quantity are indicated. My patient, Case II., felt that he knew when an attack was coming on by the increased hyperacidity of his urine, and at once began to take alkalithia, which he thought lessened the frequency and mitigated the severity of his attacks, though it did not prevent them.

It would seem to be a sensible thing to treat and remove, if possible, between attacks any local disease that may exist in the throat or upper air passages, with a view to removing the localization of the acute circumscribed œdemas in these areas. It is doubtful if this would accomplish such a highly desirable result, but it certainly should be attempted.

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MILK AND SCARLATINA.

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MILK-BORNE epidemics of scarlet fever have been reported with more or less frequency ever since the English, during the latter half of the last century, called the attention of the medical profession to this method of spreading the scarlet-fever contagion. Such epidemics have been traced in some instances to the exposure of milk to infection from scarlet-fever patients, in others to milk from cows suffering from a disease supposedly analogous to scarlet fever in human beings. From the first the English have been the ones to lay the greatest stress on the agency of milk in carrying infection, as can be seen from the fact that of 330 epidemics of infectious disease, traced to contaminated milk, no less than 243 have been recorded by English authors (Kober).[1] The usual reason given for this preponderance of milk-borne epidemics in England is that Anglo-Saxons are the only people who drink raw milk to any extent; other European peoples habitually heat or boil their milk (Freeman,[2] Kober, Hall,[3] Jürgensen[4]). It is true that America has reported only 52 epidemics as against the 243 of England, but this is said to be due to laxness of sanitary supervision of the milk supply in this country, and lack of careful search for causes in the presence of epidemics (Freeman). As further proof of the agency of milk

in disseminating scarlet fever, many authors point to the fact that countries in which milk is not used as an article of diet, or very sparingly used by children, are free from scarlet fever. Thus the disease is practically unknown among the Japanese and very rare among the Hindoos.

In reading the literature on this subject one is impressed with the difference in the attitude of Continental and of British writers. While the former admit that apparently milk is a good culture medium for the virus of scarlet fever, and that there is evidence of infection having been conveyed from one human being to another through this agency, yet their attitude toward specific instances of such milk-borne epidemics is critical, if not skeptical. They insist that before charging the milk with the responsibility of carrying the contagion, other more usual methods must be ruled out, and there must be clear proof of the relation of the milk to the spread of the disease. The responsibility of milk in the spread of scarlet fever cannot be regarded as proved beyond reasonable doubt, unless the outbreak in question presents the following features:

The epidemic must be either distinctly localized or in scattered but sharply defined foci. It must be sudden in its origin and subsidence. The persons affected in the early days of the epidemic must be drinkers of milk from a certain supply. Attendance at the same school, play-grounds, Sunday-school, etc., must be excluded. It must be shown that the milk was exposed to the contagion of scarlet fever, and the epidemic should subside after the supply of infected milk is cut off. Direct sources of infection should be ruled out, as when disease is carried by the boy who delivers the milk, or when the buyers of the milk come in direct contact with the patient in the shop.

Very few reports have proof of this character, most of them failing to exclude sources of contagion other than the milk supply. Harrington[5] gives an interesting illustration of the erroneous conclusions which may be arrived at by insufficient probing of the evidence. In an outlying part of Boston, in 1897, a sudden epidemic of scarlet fever broke out among the children of well-to-do families. Nearly all the families took milk from the same dealer. No fault could be found with the dairy farm; the cows were healthy, and there was no scarlet fever among the dairy workers. The fact, however, that all of the cases appeared in families using the same milk pointed strongly to this as the source of the contagion. Had the inquiry stopped there this epidemic would have been added to the long list of milk-borne epidemics, but a more careful search revealed the fact that the first case of all appeared in a family which had milk from another source; that this child had played with and infected other children, who had spread the disease still farther. The children belonged to the same social set and were playmates. "Thus, what might have served as a most useful example of a milk-borne epidemic fell to the ground."

As an instance of a carefully studied and critically framed report may be quoted Tingvall's[6] account of an epidemic in Westeras, Sweden. During 1901 there were only 27 cases of scarlet fever throughout the whole city. In January, 1902, within four days, 27 cases developed in 22 families living in the same part of the city. No case of scarlet fever had occurred in this region in recent times. The stricken families had nothing in common except the source of their milk supply. Eighteen families bought milk from a dairy farm almost daily; three more bought it occasionally, and almost all drank it uncooked. The one remaining family had bought milk from a farm where there had been scarlet fever shortly before. Two cases of scarlet fever were discovered in the house where the employés of the dairy farm lived. After January only 11 cases of scarlet fever developed in the whole city during the rest of the year, and none in that part of the city.

Another instance, reported as early as 1870 by Oscar Home Bell,[7] leaves nothing to be desired in the way of evidence. He describes an outbreak of scarlet fever which appeared first at St. Andrew's University; then, after the university closed for the holidays, and the milk formerly supplied to it was distributed in the town, the disease appeared in the town. The wife of the dairy farmer had a sore throat and two of her children had typical scarlet fever. She nursed them, and, at the same time, milked the cows and handled the milk. The households in which the cases occurred were, with one exception, houses supplied from this farm, and all the households taking the milk had scarlet fever except one, in which there were no children. Two boys, who had already had scarlet fever, called at the farm regularly for milk and carried it home, as a result of which three other children in the family, who had not visited at the farm, took scarlet fever.

Another careful report is that of the Rostock epidemic given by Jürgensen,[4] who is able to state that those drinking the milk boiled escaped infection. Certain of those cited by Freeman[2] are also satisfactory, as are some in Kober's[1] list, many in Hart's[8] first tabulation, some in his second,[9] Wende's[10] account of the recent Buffalo epidemic, and Eckholm's[11] account of an epidemic in Wasa.

On the other hand, the English journals are full of reports which ignore ordinary rules of evidence, and which incline one to suspect that the theory of the agency of milk in disseminating scarlet fever has taken so strong a hold upon that country that, in the absence of direct proof to the contrary, an English health officer is apt to assume that any localized epidemic of this disease is traceable to contaminated milk. The pages of the *Lancet*, of the *British Medical Journal*, and of *Public Health* abound in such reports as the following: "An outbreak of scarlet fever in Clapton. Dr. J. King Warry was able to associate the outbreak with the distribution of a particular milk supply, but he could find no evidence as to the manner

in which the milk became infected. There was no reason for thinking that the milk was infected from a human source."[12]

"Mr. Henry Armstrong has a careful report of an outbreak of scarlatina, apparently associated with the use of milk from a certain dairy. Of the 19 cases 17 took milk from the same dairy. There was some suspicion of scarlet fever in the family of a dairy worker some few months before the outbreak, but there were no other cases among the 219 other families taking the same milk."[13]

"Dr. Wallace regards 8 out of 26 cases in October as clearly connected with this contaminated milk, and 4 out of 21 cases in November as unquestionably due to the same cause." This cause was "unrecognized cases of scarlet fever in the person of the milk dealer and his child," who had suffered from "cold and sore throat." The cases were not treated medically.[14]

"In one street 21 families (more than half those supplied with the milk) yielded 29 cases; some streets taking the milk suffered no attacks. The active principle of contagion was therefore apparently not evenly distributed."[15] "Exciting cause of epidemic: Illness, suggestive of scarlatina, in the families of the two milkmen supplying the implicated milk, upward of three months prior to the outbreak."[16] "Epidemic sudden and cessation well marked; of the 205 households invaded, 74, or 34 per cent., were supplied with milk from one farm. Exciting cause of outbreak not ascertained."[17] "Scarlatina, which had hitherto chiefly affected the poorer quarters, suddenly blazed out in better-class houses exclusively, and three-quarters of the homes invaded dealt with one dairy. Cows in a febrile condition, due doubtless to the unsanitary character of their housing accommodations. A sample of mixed milk of several cows showed bacteria (character not given)."[18]

In strong contrast to this attitude are the unenthusiastic utterances of German and of some American writers:

"Observations have recently been made which indicate that it (the scarlet-fever contagion) may be conveyed in milk" (Osler[19]). "The possibility is not excluded that scarlatina can be transmitted by means of the milk" (Jürgensen[4]). "In England it has been thought that the contagion may be carried by milk" (Strümpell[20]). "Without examining into the trustworthiness of the published reports of epidemics of scarlet fever attributed to the milk supply, I would simply state that it is extremely difficult to produce indubitable proof of such occurrences so long as we are in ignorance of the specific cause of the disease. However, the mere possibility that so malignant a disease may be communicated in this way deserves our full attention" (Eckholm[11]). "The question should not be regarded as definitely disposed of, but is one that merits the most searching investigation by pathologists and bacteriologists" (Kober[21]).

This difference between English observers and those of other countries cannot be explained simply on the ground that milk-

borne epidemics of scarlet fever are more common in England than in other countries. Undoubtedly, the fact that the English drink their milk unboiled explains why there should be a greater number of milk-borne epidemics of infectious diseases there than in Germany, France, Austria, or Italy, but does not explain the enormous difference between their record and that of the Scandinavians, who also use unboiled milk. Kober[1] gives the list for Scandinavia as 11, as against the 243 of England, and Eckholm[11] speaks of the milk-borne epidemic of scarlet fever at Wasa, in 1903, as only the third thus far reported in Sweden.

In all the epidemics thus far considered the contagion is supposed to have passed from a case of human scarlet fever to the milk, and to have been carried by the milk to a healthy individual, who thereupon contracted the disease. Admitting this to be not only a possible but a fairly frequent mode of infection in scarlet fever, what shall we say as to that second class of milk-borne epidemics—those, namely, in which the disease is supposed to originate in the cow, and the contagious virus to be present in the milk at the time of milking?

The English are practically the only adherents of the theory that scarlet fever is a disease of cattle and can be conveyed from them to human beings. Their literature is full of references to outbreaks of scarlet fever and diphtheria originating in the milk of diseased cows. The first epidemic attributed to "what we now confidently recognize as scarlatina in the cow" (Hart[9]) broke out in 1885, and an investigation, ordered by the London Local Government, was carried out by W. H. Power, Klein, Cameron, and Wynter-Blythe.[22] As a result, the disease was found to be limited to the users of the milk from a dairy farm at Hendon. W. H. Power was convinced that the milk drunk by these scarlet-fever patients came from certain cows which were suffering from a vesicular and ulcerative eruption of the udder. This eruption was directly communicable from the cows to the hands of the milkers, and from them again to other cows. Klein isolated from the vesicles of both cows and milkers a streptococcus which, when injected into calves, produced in them visceral lesions resembling those of scarlet fever in man, namely, congestion of the lungs, parenchymatous degeneration and congestion of the kidneys, and hemorrhages in the spleen. He also isolated the streptococcus from the milk of the diseased cows, and from the blood of 4 out of 11 cases of scarlet fever, which streptococci he used to produce a local vesicular eruption in calves. Therefore, he concluded that this organism is the cause of scarlet fever in man, and of this particular ulcerative eruption in cows. Cameron gave a detailed description of the disease among the Hendon cows, calling it a specific, contagious, constitutional disease, capable of being communicated to man. A milker received the virus into a recent scratch on the forefinger. General weakness, malaise, loss

of appetite resulted, and, after about four or five days, a vesicle or small blister appeared on the finger; later, several on the back of the hand. The whole hand became swollen and inflamed, the inflammation extending in broad lines as far as the elbow. The general disturbance lasted a fortnight. Cameron considered the disease quite distinct from cowpox. Calves inoculated with scrapings from the ulcers developed vesicles at the site of inoculation.

The report of Power, Klein, Cameron, and Wynter-Blythe aroused much comment, and from this moment we find the attention of British physicians turned to the now famous "Hendon cow disease" as a frequent cause of epidemics of scarlet fever in man. The literature of epidemics originating in the use of milk from diseased cows increases rapidly, and it soon becomes unnecessary to show that the disease in question has the characteristics of the "Hendon cow disease." It is sufficient to state that the cows were suffering from an "obscure febrile condition;"[23] that they were "off their feed, giving ropy milk, and had scales on the teats"[24] or "abraded teats,"[25] or a rash over the body consisting of "small, red pimples."[26] Excrementitious matter or vaginal discharge may also cause contamination of the milk by the virus of scarlet fever. Thus, Power, quoted by Hart,[9] finds the origin of certain cases of scarlet fever in the use of milk from a cow who had "lost portions of her coat," and whose "buttocks and posterior udder were fouled by excrementitious matter and, perhaps, by vaginal discharge as well." Sewage-polluted water drunk by the cows, or used to wash milk-cans, is also supposed to be responsible for scarlet fever in the drinkers of the milk. Scarlyn Wilson[27] considers the discovery of "bacteria" in milk as confirmatory proof that the milk of cows kept in filthy surroundings caused scarlet fever in children, and the *British Medical Journal* makes respectful editorial comment on his report. The enthusiasm for this theory of the direct causative relation between a diseased condition in cows and scarlet fever in drinkers of their milk becomes so great that, in 1897, we find W. H. Power and Ernest Hart[9] not only accepting all the reports up to date, but adding many still more wonderful. Thus, Hart discusses the question whether a certain epidemic of scarlet fever was caused by the rinsing of milk-cans in sewage-contaminated water or by the use of milk from a cow newly calved, and inclines to the latter belief. The fact that the milk of newly calved cows can cause scarlet fever is stated by Hart to be "an important discovery of modern times," due largely to the researches of Power. Apparently the same kind of milk can cause different diseases. Thus, "ropy milk" caused an outbreak of diphtheria in Hendon in 1883, of scarlatina in Charlton in 1892, and of typhoid fever at Warminster in 1895. It is distinctly stated by Hart that the disease among the cows at Glasgow, which was supposed by Russell to be responsible for an outbreak of diphtheria in that place, was the same as the "Camberwell cow malady,"

which was considered by Sir George Buchanan and by Klein to be the cause of an epidemic of scarlet fever. The milk of newly calved cows caused scarlatina in London in 1883, at Macclesfield in 1889, and at Sutton Coldfield in 1891; while at York Town and Camberley in 1886 the same sort of milk caused diphtheria, and in Dundee in 1889 it was the "vera causa of infective quality of milk causing typhoid fever." Ernest Hart cites all these reports and concludes: "Thus, we are face to face with a fact, and it is the more important when we remember that the condition attaching to newly calved cows is not one which has been deemed of any importance by veterinary inspectors. Indeed, we may be pardoned for saying that the subject of milk-borne disease is furnishing a history of long struggle by medical as opposed to veterinary opinion. But this struggle has been harder in the matter of eruptive diseases of cows than on any other point, and the victory has certainly been with the medical experts."

Nor are these extreme views confined to one or two writers. In 1896 we find Thorne-Thorne[28] stating that the milk from diseased cows can communicate scarlet fever to man. Editorials in the *Lancet*, as late as 1892, add their testimony to the same effect, and so do the editorials in the *British Medical Journal*, as late as 1894.

Not all of the British authors, however, and practically no one in countries outside of England, hold the views outlined above. (See Allbutt's *System*, vol. iii. p. 163.) At the time of the government investigation of the famous Marylebone epidemic there were voices raised in dissent to the conclusions reached by Klein and Power, but the dissenters, being chiefly veterinarians, did not command much respect from the medical profession. The Agricultural Department of the Privy Council was roused by the statements of Power and his colleagues to make a searching inquiry into the disease among the Hendon cows, which was said to have caused scarlet fever in man. They entrusted the inquiry to Edgar Crookshank,[29] whose report is careful and convincing, and shows a singularly modern attitude toward the bacteriological aspect of the question. Crookshank had an opportunity to investigate an epidemic among cows which occurred in another county, but which was apparently the same as that just previously seen at Hendon, corresponding in every respect to the description of the latter and being communicable to calves and to milkers by direct inoculation. From the lesions on the cows' udders and on the hands of the milkers he isolated Klein's streptococcus. But among the 1500 to 2000 drinkers of the milk from this herd there was not one case of scarlatina, and neither here nor at Hendon did the disease which the milkers contracted resemble scarlet fever. He studied the lesions on the hands of the milkers and concluded that they were those of "typical Jennerian cowpox," and that the irritation and abrasion in the process of milking was responsible for the

difference in appearance of the ulcers on the cows from normal pocks. Lymph taken from the human vesicles caused typical vaccinia in calves.

Crookshank concluded that Klein's streptococcus is identical with that found in diphtheria, erysipelas, puerperal fever, scarlatina, etc., and that when inoculated into animals it sets up simply a septic inflammation, not a specific infectious disease. The lesions produced experimentally by Klein pointed only to sepsis, not to scarlatina. When this streptococcus is found in the blood of scarlet-fever patients it should be regarded as a complication, not as causally related to the scarlet fever. Other veterinarians, as Brown and MacFadyen, concurred in this report. Brown[30] stated that a disease, identical in the opinion of veterinarians with that at Hendon, prevailed in parts of eleven counties in which no scarlet fever was found. The outbreak at Hendon was, therefore, not an isolated one, and was elsewhere not associated with scarlet fever in man.

In Germany and in America the general opinion held is that the "Hendon cow disease" was cowpox, or something closely allied to it, and that the scarlet-fever epidemic was only coincident, or that it was not true scarlet fever, but a septic fever due to the presence of pus in the milk. Careful examination of the reports, unfortunately often very meagre, of cases of scarlet fever caused by the drinking of milk from diseased cows brings to light certain features which suggest a disease other than typical scarlatina. Baginsky,[31] for instance, comments on the mild character of the disease in the Hendon epidemic, only 1 fatal case among 60, and the short incubation period of two days. The statement as to a one-to two-day incubation period is also made by C. N. Cooper.[32] Clothier,[33] in his account of the outbreak at Stroud Green, says: "As has been observed in previous epidemics of scarlatina disseminated by milk, the type of the disease is mild, and no deaths are reported."

Harrington[34] abstracts the report of an epidemic in Lincoln, England, in 1903, involving 40 persons, the majority of whom were seized on the same day and none of whom was seriously ill. There was a remarkable susceptibility to infection in this epidemic, no less than 14 persons in one household suffering from the disease, a fact which certainly would in itself incline one to doubt the diagnosis of scarlet fever. The milk to which this attack was attributed came from cows with vesicles on the udders. In an epidemic recorded by G. Buchanan[35] the persons attacked with what was held to be scarlet fever, 19 in number, were all adults. Cream of which all had partaken was held responsible for the outbreak, but there was no evidence that any case of scarlet fever had come in contact with it.

Now, if we assume that the milk in these instances was infected, not with the specific virus of scarlatina, but with pyogenic cocci from the inflamed udders of the cows, the matter becomes clear at once. That mammitis in the cow is accompanied by the appear-

ance of streptococci and staphylococci in the milk has been proved by many investigators. Guillebeau[36] examined the milk of 76 cows with inflammation of the udder and found that all contained pyogenic cocci virulent for animals. Nocard and Mollerau[37] demonstrated streptococci in the milk of cows with mammitis, as did also Adametz,[38] Hueppe[39] and others, to whose researches must be added those of Klein, cited above.[29] Bergey[40] found that streptococci and staphylococci persist in the milk for some time after all external signs of mammitis have subsided. At the height of the inflammation these germs are present in enormous numbers. The descriptions of the condition of the cows in the Hendon epidemic, and in others resembling it, show that pus, scales, and blood must have found their way into the milk through the friction of the milkers' hands. Dr. Russell[41] found cocci in the milk of a cow suffering from the Hendon disease, and also in the blood of the calf she was suckling. In other cases it is apparent that the cows were suffering from a catarrhal mammitis at the time, or shortly before the epidemic among drinkers of the milk. The theory of Power as to the infectious nature of the milk of newly calved cows may have some foundation in the greater tendency to mammitis just after calving, or possibly the cows may have been suffering from a mild form of puerperal sepsis.

There are, in fact, two or three reports of cases of throat inflammations attended with constitutional symptoms, which apparently came from the drinking of milk containing pus. Kober[21] describes two epidemics of follicular tonsillitis in a boys' school, with severe gastric disturbances; one involving 50 and the other 84 cases. The cows showed the lesions of variola vaccinia. In 1869 Fagan[42] reported a case of pseudomembranous stomatitis following the drinking of milk contaminated with pus and blood from an abscess in the udder of the cow. Gray Edwards[43] describes with much care and accuracy some cases of follicular tonsillitis due to infected milk. Cultures from the throats of the 12 cases and from the milk yielded the same organisms, namely, staphylococcus aureus and albus and streptococcus. Three children who drank only boiled milk escaped the infection. The "ropy," or "stringy," or "slimy" condition of the milk mentioned by some authors, and especially emphasized by Power and Klein as the cause of scarlet fever and diphtheria, is a condition caused by a wide variety of organisms, including certain strains of staphylococcus pyogenes (Passet, Lehman, and Neumann), and various other cocci and bacilli.*

* Bac. lactis sapon. (Weigmann and Zirn, Centralbl. f. Bakt., Bd. xv. p. 463); Micrococcus Freudenreichii and Bacterium Hessii (Guillebeau, Centralbl. f. Bakt., 1892, Bd. xi. p. 439); Bacillus lactis viscosus (Adametz, Centralbl. f. Bakt., 1891, Bd. ix. p. 698); Leischmann's bacillus (Centralbl. f. Bakt., Bd. xvi. p. 122). Reference to still others is made in Adametz's original article (Berliner Landw. Jahrb., 1891), cited above. Guillebeau's micrococcus Freudenreichii is considered by Lehmann and Neumann as probably a streptococcus. Neither his nor Adametz's organisms were pathogenic.

It is evident that milk-borne epidemics of scarlet fever do occur, but there seems no reasonable basis for the belief that they can originate in a diseased condition of the cow. In this connection we cannot do better than quote the words of Kober.[21] "When we consider that toxins may and do produce a scarlatinous exanthema, we feel warranted in saying that in all the epidemics of scarlet fever and diphtheria which were traced to milk from cows suffering from inflammatory lesions of the udder or from puerperal fever we have typical instances of a streptococcus and staphylococcus infection. . . . There is no proof that there is a disease in the cow which is communicable as scarlet fever to man."

In view of our ignorance as to the infectious agent in scarlet fever, it is interesting to know that milk serves as a good culture medium for it. Most authentic reports of this kind show that the milk was directly exposed to the contagion, as when the milking or handling of the milk or washing of the cows was carried on by persons suffering from a mild form of scarlet fever, or in the stage of desquamation following it. In many instances, however, the contact is less direct; a healthy person apparently carries the infection from a case of scarlet fever to the milk. Wives and daughters of dairymen nurse cases of scarlet fever and handle the milk at the same time. Some of these reports must undoubtedly be taken with caution, for the possibility is not excluded that the selling of the milk was carried on in the room where the sick person lay, or the delivery made by a person ill with scarlet fever, but in others the evidence of indirect infection is very clear. Of Hart's 99 epidemics of milk-borne scarlet fever, the disease was said to exist either at the dairy or on the milk farm in 68. In 17 of these, persons actually suffering from or recovering from scarlet fever handled the milk, but in others the connection was less direct. Two were supposedly caused by bottles or cans left in infected houses; 1 by the wiping of cans with an infected cloth; while in 3 the milk, though not handled by the patient, was stored in or close to the sick-room. In 6 instances dairy employés lodged in or had visited infected houses. Whittaker[44] gives an instance of infection of the milk and of the milkman's daughter through rags stored in the house and used to wipe the cans. The girl came down with the disease in another town on the day following her departure from home.

Many authorities on scarlet fever treat with a certain suspicion the instances given in the literature of infection through a third person, himself free from the disease. As the scarlet-fever virus is not highly infectious, and is apparently confined to the air immediately around the patient, many are inclined to believe that the carrying of the infection by third persons is very rare. But if, as seems very probable, milk is a good culture medium for this virus, it is easy to see why it should figure prominently in such indirect

infection. Kerschensteiner[45] says that if infection is to take place through a healthy third person the "scarlet fever dust" must be blown to that person, must cling to his skin, hair, or clothes, and must be blown off from him to the third person; and as scarlet fever is not very infectious, a large amount of the infectious stuff is needed. Now, if we imagine the virus not carried in a dry condition and inhaled as dust, but planted in a liquid culture medium and then, after a lapse of time sufficient for its growth, brought in contact with the throat in the process of swallowing, it is easy to believe that infection through the medium of a healthy third person can take place with comparative frequency, and also that utensils exposed to the air of the sick-room, even at a distance from the patient, may become infected sufficiently to carry the disease.

CONCLUSIONS. The literature of milk-borne epidemics of scarlet fever contains many which are (*a*) based on insufficient evidence as to the agency of milk in the dissemination of the disease; (*b*) based on the erroneous belief that a disease in cows is capable of causing scarlet fever in man.

The disease in cows supposedly responsible for scarlet fever in man is ordinary cowpox, and the disease in man supposed to be caused by it is either septic fever from infection from pyogenic organisms or coincident scarlet fever.

There is no good evidence that milk from diseased cows can cause scarlet fever in man.

After rejecting many reports of milk-borne outbreaks of scarlatina there remains a fair number which are above criticism, and which apparently prove that milk is a good culture medium for the scarlet fever virus, and is the most frequent agent of indirect infection in this disease.

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THE CAUSE OF PULSATIONS IN EMPYEMA.

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GUILLIAM DE BAILLON in the sixteenth century reported the first authentic case of pulsating empyema.

Le Roy (1776) speaks of the possibility of pulsating empyema in his Aphorisms, 489: "When the pus sac lies on the heart and large vessels in such a manner as to transmit their movements there develop false impressions of an aneurysm."

Berard (1836), Stokes (1838, first used the term pulsating empyema), MacDonnell (1844, first used the term empyema necessitatis pulsans), Walshe (1851), Wintrich (1854), Flint (1856), Aran Heyfelder, Owen Ress (1858), Schoostek (1865), Gourbon (1870), and Muller Plagge, Traube (1872), Frentzel (1875), Topham and Dunin (1878) each report one case.

Traube thinks the pulsations are due to a pericardial effusion associated with the empyema.

Comby (1881), reviewing the literature, reported a case and explained the pulsation by saying: "When the lung is completely collapsed, pushed to one side and adherent to the pericardium, it no longer resists the heart, the pulsations of which are then transmitted to the fluid in the pleural cavity, thence to the thoracic wall."

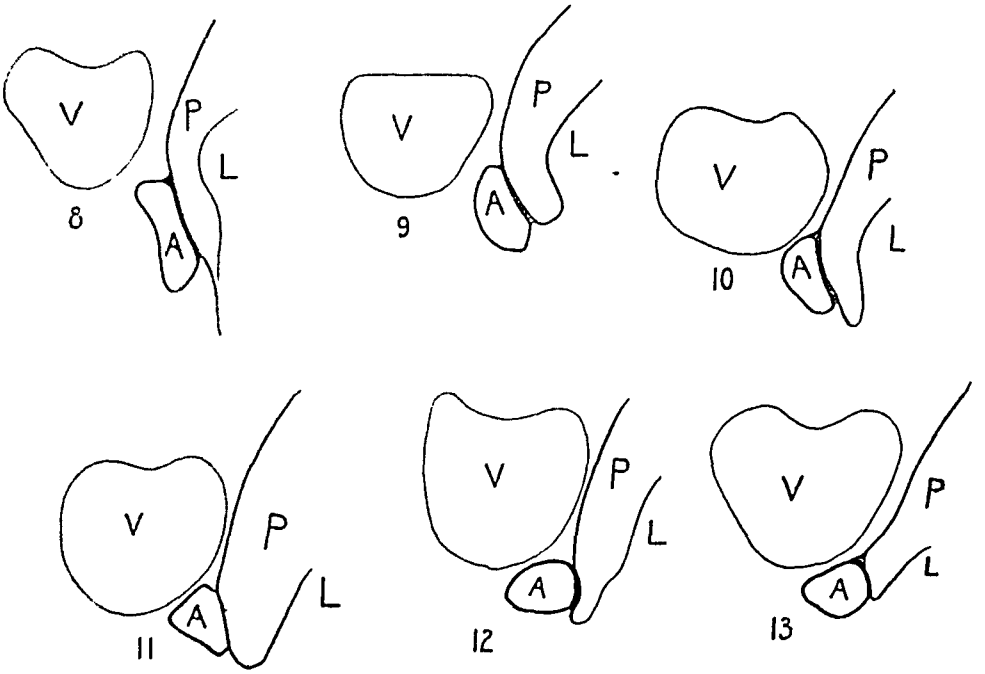
Fercol (1884) reported a case of pyopneumothorax pulsans and says the pulsations are due to the presence of the air.

Skoda Gutbrod says, "In systole the heart has a right to left movement which may explain the pulsation."

Frantzel contradicts Traube's theory that a pericardial exudate is necessary to cause pulsations.

Kussmal says, "When the pleural wall next the heart is thick enough to resist the impulse of the heart, the heart beat is transmitted through the pleural wall to the fluid."

The recent literature, at my disposal, adds no new theories to account for the pulsations, and in each the author states that the theories advanced are inadequate.



The relationship of the aorta to the parietal pleura in six transverse sections of the thorax is shown. Sections of the trunk are numbers eight, nine, ten, eleven, twelve, and thirteen. *V*, vertebra; *A*, aorta; *P*, pleural cavity; *L*, lung. Section eight is on a level with the upper border of the sixth vertebra; nine, with seventh vertebra; ten, with eighth vertebra; eleven, with ninth vertebra; twelve, with tenth vertebra; and thirteen with eleventh vertebra. From the point where the aorta touches the vertebra until it enters the diaphragm it is in immediate contact with the parietal pleura.

At the level of sections number nine, ten, eleven, twelve, and thirteen the right parietal pleura could easily be forced against the aorta.

While working on cross-sections of a body with pleurisy of the left side, the anatomical relationship of the organs suggested to me an explanation for the pulsating empyema which on analysis seems to fulfil all the requirements.

The requirements are a firmly fixed pulsating organ; contact of the pleural wall with this pulsating organ; distention of the pleural sac with fluid, air, or solid material, and collapsed condition

of the lung. The first requirement is fulfilled by the thoracic aorta; the second, by the normal relationship of pleural wall to thoracic aorta; the third, by presence of fluid, pus, or a combination of these with air in the pleural cavity; and the fourth, by the collapsed condition of the lung in pleurisy and empyema.

The thoracic aorta above lies slightly to the left of the median line on the bodies of the vertebra. In the lower part of the thorax it is almost in the median line. Immediately over the walls of the aorta is the parietal pleura. This relationship continues from the fourth to fifth rib above as far down as the diaphragm. The aorta is firmly anchored by the intercostal arteries, so that it requires considerable force to move it either to the right or left. This proximity of the aorta to the parietal pleura is well shown at *A* and *P* in the figure.

In the normal thorax the pulsations of the aorta are absorbed by the easily compressible lung tissues, consequently are not transmitted. When fluid is present in the left pleural cavity the lung is partially or completely collapsed, depending on the amount of fluid. As the fluid increases the thoracic walls and mediastinum are pushed outward from the cavity of the left pleura and the lung becomes more and more collapsed. Until the lung is completely collapsed it naturally contains more or less air, consequently compressible and capable of absorbing, or compensating for, the impulse of the aorta. But in time a point is reached when to further compress the lung a pressure sufficient to further expand the thoracic wall, more especially the intercostal spaces, is required. Now, the impulse of the aorta transmitted to the pleural effusion will further expand the intercostal spaces or pulsation of the intercostal spaces is present.

The pressure in the pleural cavity is transmitted in all directions at right angles to the pleural walls, consequently the aorta is pushed to the right as far as the left intercostal arteries permit and against the vertebral column. The arterial wall expands in all directions. The artery being firmly fixed against the vertebral column and nearly half-covered by the parietal pleura, which does not offer a resistance equal to that offered by the vertebral column and stretched left intercostal arteries, the aortic pulsations must be transmitted to the fluid in the pleural cavity. This pulsation of the effusion is transmitted equally in all directions and must be taken up by the elasticity of the mediastinal membranes, diaphragm, and thoracic wall and the compressibility of the lung. Naturally, of these structures the one with the greatest elasticity will absorb the greatest portion of the impulse. Normally, the lung is sufficiently elastic, consequently sufficiently compressible, to absorb the entire impulse; but when collapsed and solid its elasticity is much less than that of the thoracic wall, which must pulsate synchronously

with the aorta, or systolic in time. If the thoracic wall is weaker than normal, due to inflammation and degeneration of the muscles and parietal pleura or to paralysis of the intercostal muscles, the pulsations naturally will occur under a lower intrapleural pressure than when the thoracic wall is normal; or pulsation of the thoracic wall will occur in pleural effusions whenever the elasticity of the lung becomes less than that of the thoracic wall—in other words, when it requires more pressure to further compress the lung than it requires to further expand the thoracic wall. The mediastinal membranes and diaphragm probably pulsate to some extent. But the pulsation of the one is absorbed by the normal lung, and of the other, by the abdominal organs.

As this pulsation is purely hydraulic in principle, the nature of the fluid in the pleural cavity is of little or no importance. Consequently in pleural effusions pulsation is not a positive sign of empyema. In the literature at hand only three cases of pulsating pleurisy are recorded.

When a part of the fluid is withdrawn a cessation of pulsation has been recorded. The withdrawal of the fluid reduces the intrapleural pressure. The lung expands when its elasticity becomes greater than that of the thoracic wall. Consequently the lung again absorbs the aortic pulsation.

The majority of cases of pulsating empyema occur on the left side. Only a few cases in the right side are recorded. An examination of cross-sections of the thorax shows that the right parietal pleura may easily be pushed against the aorta so that its pulsations may be transmitted to fluid in the right pleural cavity.

I am indebted to Dr. C. M. Jackson, Professor of Anatomy, University of Missouri, for the transverse sections of the trunk.

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AN INVESTIGATION OF THE INFLUENCE OF ADRENALIN CHLORIDE ON TOXIC DOSES OF COCAINE.[1]

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THE danger associated with the use of general narcosis has led to the employment of numerous drugs and methods of admin-

[1] Read at the Annual Meeting of the Second District Branch of the New York State Medical Association, held at Saratoga in May, 1905.

istration in the hope of finding a perfect local anæsthetic. On this account, the introduction of cocaine by Koller met with ready acceptance; and the discovery of spinal anæsthesia in 1884 by Corning (rediscovered and extensively used by Bier) gave an additional stimulus to the use of cocaine.

The great disadvantage associated with the use of a local anæsthetic is the danger to the individual resulting from the absorption of the drug used as the anæsthetic. Cocaine is such a drug. It is almost perfect in its local application, making possible the severest operations free from pain, and yet at times cocaine poisoning may result from the absorption of the drug. Various methods have been employed in the endeavor to prevent the occurrence of this toxic action. Since the toxicity of cocaine presumably comes from its entrance into the blood the first need was a knowledge of a means of hindering its absorption.

One of the first methods used to accomplish this was that of Reclus and Schleich, with the introduction of what is known as Schleich's solution. The anæsthesia depends upon the bulk of the fluid. Large quantities of a weak solution of cocaine are injected and thus only a small amount of cocaine comes into contact with the tissue, and the danger of absorption is thus minimized. The chief objection to the use of Schleich's solution is the œdema produced and the consequent loss of normal appearance of the tissue. The constriction of the circulation above the part to be anæsthetized, in order to prevent absorption, has marked limitations. The use of an ethyl chloride spray to cool the part previous to the injection of the cocaine, as introduced by Braun, has not been altogether satisfactory. He has demonstrated that, so long as the vitality of tissue is kept lowered by cooling, absorption is hindered.

Klapp experimented on dogs with an oily solution of cocaine. He claims that an oily solution of cocaine prevents the capillaries and lymphatics from taking up the cocaine as rapidly as they otherwise would and by this means it prevents the toxic symptoms resulting from the absorption. Glycerin is found to have a similar action in retarding absorption.

The latest and most universal method for the reduction of the toxicity of cocaine is its use in combination with adrenalin chloride. Scarcely a medical journal can be perused without finding some reference to this new anæsthetic.

In the *Archiv f. klin. Chir.*, Bd. lxix., Braun gave the results of his experiments with combinations of adrenalin and cocaine. He employed rabbits for his experiments, giving subcutaneous injections of adrenalin chloride followed ten minutes later by similar injections of cocaine. Previous determination had shown that 0.1 gm. cocaine per 1000 gm. of body weight was a fatal dose. Two typical experiments are here given:

1. Rabbit weighing 1300 gm. injected subcutaneously with 1 c.c. of a 1 : 1000 adrenalin chloride solution. Ten minutes later 0.13 gm. cocaine injected subcutaneously into the same place. In five minutes paralysis, no convulsions. Animal recovered.

2. Rabbit weighing 1700 gm. injected subcutaneously in the head with 1 c.c. normal salt solution and with 1 c.c. of 1 : 1000 adrenalin chloride subcutaneously in the back. Ten minutes later 0.17 gm. cocaine injected subcutaneously in the head in the same place as the salt solution. In five minutes paralysis, convulsions. Animal recovered.

From these and similar experiments the conclusion was reached that previous injections of adrenalin inhibit the toxic action of cocaine. The inhibitory action also takes place, but to a less degree, when the injection of the two drugs is not made in the same place.

In a subsequent article (*Centralblatt f. Chir.*, Bd. xxx., No. 38), Braun confirmed his previous assertion as to the value of combining adrenalin and cocaine. In a third paper (*Archiv f. klin. Chir.*, Bd. lxxi.) he stated that the adrenalin cocaine mixture as compared to cocaine from the standpoint of an anæsthetic is quicker, stronger, lasts longer, and is less toxic.

Foisy in using a mixture of cocaine and adrenalin as compared to cocaine alone found:

1. Greater anæsthetic effect.
2. Greater local vasoconstrictor effect.
3. Less toxicity.

He mentioned two sets of experiments. The first set of experiments was on man with a mixture as follows:

Chlorohydrate of cocaine 1 : 200	10 c.c.
Adrenalin chloride 1 : 1000	10 drops.

This mixture was employed in 149 diseases in doses from 2 to 10 c.c. without experiencing the accident of cocaine intoxication. In giving the same doses of cocaine without adrenalin toxic effects, such as paleness, sweating, talkativeness, muscular tremor, and delirium frequently developed.

The second set of experiments was with subcutaneous injections in animals. The toxic dose of cocaine according to Laborde is 0.01 gm. per 1000 gm. body weight. With mixtures of adrenalin and cocaine in this dosage Foisy has never produced contractions or convulsions in animals.

Donitz reported a large number of experiments confirming Braun's findings. Cats were injected by subdural lumbar puncture and it was found that by injecting adrenalin and cocaine together the toxicity of the latter could be reduced one-third while injections of the adrenalin six minutes before the cocaine reduced the toxicity one-fifth.

Braun's findings were that: (1) adrenalin lessens the toxicity of cocaine; (2) it enhances its anæsthetic strength for duration, extent,

and intensity. Donitz finds that both these hold for subdural application.

The experiments of Ziegan as regards the amount of adrenalin chloride that can be injected without harm are of interest. He found that 0.001 gm. of adrenalin injected subdurally by lumbar puncture in cats caused anæsthesia of the hinder parts in eight to twelve minutes. If, after the injection, the animal was held with its head downward there resulted a general anæsthesia without loss of reflexes lasting thirty to fifty minutes. In two hours all effects of the drug disappeared. An injection of 0.003 gm. caused tonic convulsions in the lower extremity, and injections of 0.005 gm. killed by œdema of the lungs and paralysis of the respiration.

An interesting series of experiments was performed by Thies, the results of which were contradictory to those of Braun and Donitz. Cats were used for the experiments, nearly all the injections being made by lumbar puncture. The dosage and results are shown in the two following tables. Table A represents those cases where the adrenalin and cocaine were injected together. Each experiment has a control represented by an animal of equal weight receiving the same dose of cocaine without adrenalin admixture.

TABLE A.

0 02 C + 0 0005 A	Lived	0 02 C	Lived.
0 02 C + 0 0005 A	"	0 02 C	"
0 02 C + 0 0005 A	Died.	0 02 C	"
0 03 C + 0 0005 A	Lived	0 03 C	"
0 04 C + 0 0005 A	"	0 04 C	"
0 04 C + 0 0005 A	Died	0 04 C	"
0 05 C + 0 0005 A	"	0 05 C	"
0 05 C + 0 0005 A	Lived	0 05 C	"
0 06 C + 0 0005 A	Died.	0 06 C	"
0 08 C + 0 0005 A	"	0 08 C	Died.
0 1 C + 0 0005 A	"	0 1 C	"
0 1 C + 0 0005 A	"	0 1 C	"
0 2 C + 0 0015 A	"	0 2 C	Lived
0 2 C + 0 001 A	"	0 2 C	"
0 25 C + 0 0005 A	"	0 25 C	"

From the above table it will be seen that the amount of cocaine necessary for fatal doses was greater where cocaine was used alone than in those cases where it was used in combination with adrenalin.

Table B shows the results where the adrenalin was injected by lumbar puncture followed in six minutes by the injection of cocaine. There are three of these experiments with controls of animals of equal weight receiving the same dose of cocaine.

TABLE B.

0 06 C + 0 0005 A	Died	0 06 C + salt sol	Lived
0 06 C + 0 0005 A	"	0 08 C + salt sol	Died
0 08 C + 0 0005 A	"	0 08 C + salt sol	"

Table B shows the same results as Table A. A certain dose of cocaine (0.06 gm.) injected in combination with adrenalin is fatal,

but the same dose of cocaine when used alone is not fatal. A dose of 0.08 gm. of cocaine causes death whether injected alone or in combination with adrenalin.

Thies observed that the symptoms of cocaine poisoning did not come on so rapidly in those cases where adrenalin was used. He concluded that the toxic action of cocaine was not destroyed or diminished by adrenalin, but was simply retarded. With fatal doses of cocaine adrenalin has therefore no effect in diminishing its toxic action.

In the *Berl. Klinik* for January 1, 1904, Braun gave a summary of the work done on adrenalin cocaine anæsthesia and discussed Thies' article. His criticism of the paper is that the doses of cocaine are very large. Braun wishes to emphasize that in the adrenalin cocaine combination *smaller* doses of cocaine can be used.

That adrenalin chloride has an inhibitory action on the general absorption of fluids cannot be doubted after the recent work of Meltzer and Auer. Klapp found that one drop of a 1 : 1000 adrenalin solution injected subcutaneously into dogs' backs would hinder the absorption of milk-sugar.

In the use of cocaine as an anæsthetic the danger is from the toxic effect due to its absorption, and it is a common observation that this does occur. The toxicity is shown by such symptoms (described by Foisy) as paleness, sweating, talkativeness, muscular tremor, delirium, and even death. It would seem, therefore, that in testing the value of adding adrenalin chloride to cocaine in its use as a local anæsthetic the effect of adrenalin on *toxic* doses of cocaine should be carefully studied. The following experiments were undertaken with this in view:

Protocols.

Experiment No. 1. Guinea-pig No. 1, weighing about 500 gm., injected with 0.5 c.c. of a 10 per cent. solution of cocaine into the abdominal cavity. After four minutes retraction of head. After eight minutes convulsions. After ten minutes death.

Experiment No. 2. Guinea-pig No. 2, weighing about 500 gm., injected with 0.5 c.c. 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later 0.06 gm. cocaine injected into the abdominal cavity. After four minutes paralysis of hind legs. After eight minutes convulsions. After fourteen minutes death.

Experiment No. 3. Guinea-pig No. 3, weighing about 500 gm., injected with 0.025 gm. cocaine into the peritoneal cavity. After thirteen minutes convulsions. Animal recovered in thirty-five minutes.

Experiment No. 4. Guinea-pig No. 4, weighing about 500 gm., injected with 0.5 c.c. 1:1000 adrenalin chloride solution into the peritoneal cavity. No marked symptoms. Ten minutes later

0.025 gm. cocaine injected into peritoneal cavity. Animal died in thirty-nine minutes. No symptoms of cocaine poisoning. Animal died apparently from œdema of lungs. Symptoms came on twenty minutes after injection of adrenalin.

Experiment No. 5. Guinea-pig No. 5, weighing about 500 gm., injected with 0.5 c.c. 1:1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms.

Experiment No. 6. Guinea-pig No. 6, weighing about 1000 gm., injected with 1 c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later 0.05 gm. cocaine injected into abdominal cavity. After twenty minutes well-developed paralysis of hind legs. After forty-five minutes animal had apparently recovered, but it died during the night several hours afterward.

Experiment No. 7. Guinea-pig No. 7, weighing about 1000 gm., injected with 0.05 gm. cocaine into the abdominal cavity. After eight minutes paralysis of hind legs. After ten minutes retraction of head. After seventeen minutes convulsions. Animal recovered in one hour.

Experiment No. 8. Guinea-pig No. 5, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride in abdominal cavity. No marked symptoms.

Experiment No. 9. Guinea-pig No. 8, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride into the abdominal cavity. Ten minutes later 0.025 gm. cocaine injected into abdominal cavity. No marked prostration. No convulsions. Animal recovered in thirty minutes.

Experiment No. 10. Guinea-pig No. 9, weighing about 750 gm., injected with 0.037 gm. cocaine into abdominal cavity. After ten minutes falls on side. After twelve minutes retraction of neck. No convulsions. Animal recovered in thirty minutes.

Experiment No 11. Guinea-pig No. 10, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. Ten minutes later injected with 0.025 gm. of cocaine into the abdominal cavity. After fifteen minutes paralysis of hind legs. After twenty-five minutes animal seemed to partially recover but died suddenly, thirty-seven minutes after the injection of the cocaine.

Experiment No. 12. Guinea-pig No. 11, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. After fifteen minutes paralysis of hind legs. After eighteen minutes falls on side. After twenty-five minutes recovered somewhat, but died later.

Experiment No. 13. Guinea-pig No. 12, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride solution into

the abdominal cavity. No symptoms. Ten minutes later injected with 0.025 gm. of cocaine into the abdominal cavity. After fifteen minutes slight paralysis of hind legs. After twenty-five minutes falls on side. After twenty-seven minutes died suddenly.

Experiment No. 14. Guinea-pig No. 13, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. After fifteen minutes lies on side. After twenty-four minutes head retracted. Animal died several hours afterward.

Experiment No. 15. Guinea-pig No. 14, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. After twenty minutes paralysis of hind legs. After twenty-four minutes falls on side. Seemed to recover, but died several hours later.

Experiment No. 16. Guinea-pig No. 15, weighing about 750 gm., injected with $\frac{3}{4}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.037 gm. cocaine into the abdominal cavity. After twenty minutes paralysis of hind legs. After thirty-five minutes falls on side. After forty minutes death.

Experiment No. 17. Guinea-pig No. 16, weighing about 500 gm., injected with $\frac{1}{4}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into abdominal cavity. After fifteen minutes paralysis of hind legs. After twenty minutes falls on side. After thirty minutes retraction of neck. Animal recovered.

Experiment No. 19. Guinea-pig No. 18, weighing about 500 gm., injected with $\frac{1}{4}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. After fifteen minutes slight paralysis of hind legs. Animal seemed to recover in large measure, but died several hours after.

Experiment No. 20. Guinea-pig No. 8, weighing about 500 gm., injected with $\frac{1}{4}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. Slight paralysis of hind legs. Animal recovered.

Experiment No. 21. Guinea-pig No. 5, weighing about 500 gm., injected with 0.025 gm. cocaine into the abdominal cavity. After thirty minutes slight paralysis of hind legs. Animal recovered.

Experiment No. 22. Guinea-pig No. 3, weighing about 500 gm., injected with $\frac{1}{4}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. After

seven minutes falls on side. After twelve minutes convulsions. Animal died in twenty minutes.

Experiment No. 23. Guinea-pig No. 9, weighing about 750 gm., injected with $\frac{3}{8}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later 0.037 gm. cocaine injected into the abdominal cavity. After thirty minutes slight paralysis of hind legs. Animal recovered.

Experiment No. 24. Guinea-pig No. 5, weighing about 500 gm., injected with $\frac{1}{4}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. After thirty minutes slight paralysis of hind legs. Animal seemed to recover, but died several hours afterward.

Experiment No. 25. Guinea-pig No. 7, weighing about 1000 gm., injected with 0.05 gm. cocaine into the abdominal cavity. No symptoms. Animal recovered.

Experiment No. 26. Guinea-pig No. 8, weighing about 500 gm., injected with 0.025 gm. cocaine into the abdominal cavity. After fifteen minutes paralysis of hind legs. After twenty minutes falls on side. Animal recovered in forty minutes.

Experiment No. 27. Guinea-pig No. 16, weighing about 500 gm., injected with 0.025 gm. cocaine into the abdominal cavity. After twenty-five minutes slight paralysis of hind legs. Animal recovered.

In order that the effects of the drugs might come on quickly and be influenced easily it was deemed best to make the injections into the peritoneal cavity of guinea-pigs. The results can be better understood by separating the experiments into groups and arranging the groups into tables. In all cases the injection was made into the peritoneal cavity. When the combination of drugs was used the adrenalin was injected first, followed ten minutes later by cocaine. The dosage was in proportion to the weight of the animal, so that each animal in the various groups received comparatively the same amount of drug. The first group consists of one experiment and its control. The dosage of adrenalin was 0.001 gm. per kilo of body weight, while of cocaine it was 0.1 gm. per kilo.

TABLE I.

Experiment No.	Injection of	Onset of symptoms in	Result.	Control.			
				Experiment No.	Injection of	Onset of symptoms in	Result.
2	Ad. + Co.	4 minutes.	Died 14 min.	1	Co.	3 minutes.	Died 10 min.

The experiment showed that the dosage of cocaine was too great, but even with this overdose the retarding action of adrenalin was manifest.

In the experiments in Table II. the dose of cocaine has been reduced to 0.05 gm. per kilo, while the dose of adrenalin is the same. Control animals in which adrenalin alone was injected were used first.

TABLE II.

Experiment No.	Injection of	Symptoms began in	Result.	Control.			
				Experiment No.	Injection of	Symptoms began in	Result.
				5	Ad.	No symptoms	Recovered
				8	Ad.	"	"
4	Ad. + Co.	10 min.	Died	3	Co.	8 min.	"
6	Ad. + Co.	20 "	"	7	Co.	8 "	"
9	Ad. + Co.	No marked symptoms.	Recovered	10	Co.	10 "	"
11	Ad. + Co.	15 min.	Died	17	Co.	No symptoms.	"
12	Ad. + Co.	15 "	"				
14	Ad. + Co.	15 "	"				
15	Ad. + Co.	20 "	"				
16	Ad. + Co.	20 "	"				

The above table shows a remarkable record. Not an animal of the series which received the dose of cocaine alone died, while only one of the adrenalin-cocaine animals lived. By referring to the protocols it will be found that animals Nos. 6, 12, 14, and 15 seemed to recover in large measure, but died later. Animal No. 4 died apparently of œdema of the lungs, but it was the only animal in the whole series of experiments that showed any marked effect from the dosage of adrenalin. It will be noticed that the symptoms come on somewhat quicker in the animals that were injected with cocaine only.

In Table III. the experiments were the same as above except that the dosage of adrenalin has been reduced to 0.0005 gm. per kilo. The reduction in the amount of adrenalin was made on the supposition that, inasmuch as the animals were receiving almost a lethal dose of both adrenalin and cocaine, the combination proved fatal.

TABLE III.

Experiment No.	Injection of	Symptoms began in	Result.	Control.			
				Experiment No.	Injection of	Symptoms began in	Result.
18	Ad. + Co.	15 min.	Recovered	25	Co.	No symptoms.	Recovered
19	Ad. + Co.	15 "	Died	26	Co.	20 min.	"
20	Ad. + Co.	25 "	Recovered	27	Co.	25 "	"
21	Ad. + Co.	30 "	"				
22	Ad. + Co.	7 "	Died				
23	Ad. + Co.	30 "	Recovered				
24	Ad. + Co.	30 "	"				

Before making any conclusions from these experiments it should be noted that all of the animals except Nos. 18, 19, and 21 had had previous injections of cocaine. The possibility of a slight immunity having been established should be considered. Three out of the seven adrenalin cocaine animals died while none of the control animals died.

What then are the conclusions to be drawn? The same criticism holds here that Braun made of Thies' work. The experiments are made with too large doses of cocaine to show the real value of adrenalin cocaine as a local anæsthetic. What is certainly shown is that *adrenalin will not protect the organism against toxic doses of cocaine.*

In the use of adrenalin cocaine care should be exercised not to inject a toxic dose of the latter, for not only does the adrenalin fail to protect the body against the toxic doses of cocaine, but it seems to enhance the toxic action.

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THE INFLUENZA BACILLUS IN BRONCHIECTASIS.

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THAT dilatation of the bronchi may occur in the course of acute influenza, either of the bronchial or pneumonic type, has been demonstrated by necropsy findings and the cultivation of the specific organism from the lesions. (Leichenstern, M. Beck, and others.) Similar cases have also been reported in which recovery from the acute illness occurred with the gradual development of chronic bronchitis or bronchiectasis.

In our own country the relationship of bacillus influenzæ to chronic pulmonary disease has received but little attention, if we except the extensive investigations of Lord.

It may therefore be of interest to present the findings in some cases observed at the Johns Hopkins Hospital (service of Prof. Osler) during the early part of 1905.

CASE I. *Total duration three months; onset with symptoms of acute coryza; gradual development of cough and profuse expectoration; small hemorrhages, irregular temperature, sweats; influenza bacilli in pure culture from sputum; headache, partial paralysis; death; autopsy: diffuse bronchiectasis, bronchial erosion, patches of bronchopneumonia, small foci of necrosis; influenza bacilli in pure culture from tissues; no tuberculosis.*—B. R. F., male, white, aged twenty-nine years, trained nurse. Admitted February 16, 1905, complaining of "cough and pains in chest."

Family History. Good; negative for tuberculosis.

Past History. No acute diseases of childhood; never any pneumonia; no typhoid fever; no hemoptysis; severe la grippe three years ago; lasted two months; no other pulmonary trouble until present illness; no history of gastric or intestinal disease; no renal troubles; primary syphilitic sore five years ago; no secondaries; always strong and healthy; moderate use of alcohol; heavy smoker of cigarettes; average weight 145 pounds.

Present Illness. Two months before admission patient caught cold after exposure. This was at first slight, not accompanied by cough. It slowly grew worse, however, and there has been severe cough with increasingly profuse expectoration; for about three weeks patient has felt feverish at night. In past four days the cough much worse, with nocturnal dyspnœa, fever, sweats, and pain in right side. He has lost fifteen pounds in weight since onset.

Physical Examination. On admission patient was well nourished and not in great distress. Diminished expansion more marked on right side. Vocal fremitus impaired, with dulness on percussion over right axilla and lower back and over left lower back. Breath sounds enfeebled over dull areas, though nowhere absent. No tubular breathing. No friction rub. No movable dulness. Very numerous moist and sonorous rales over entire chest. Heart sounds clear and of normal relative intensity. Abdomen normal; deep reflexes normal. Temperature irregular, varying in first twenty-four hours from normal to 100.5°. Pulse soft, regular, 96 to minute. No marked thickening of vessel walls. Sputum profuse, greenish, mucopurulent, with a sickening, sweetish odor. Examination for elastic tissue and tubercle bacilli negative. Exploration of chest with needle yielded no results. Leukocytes on admission, 22,000; hæmoglobin, 90 per cent. Patient did not improve, paroxysms of coughing grew worse.

February 24th. First culture from sputum absolutely pure growth of bacillus influenzæ.

25th. Small hemorrhage from lungs, followed by chill and sweat. Pulmonary signs remained about as at first, except for increase in sonorous rales. Leukocytes varied between 12,000 and 25,000.

March 8th. Patient complained of numbness and some apparent weakness in right hand. Some headache. Two days later pain in

right testicle, with increased weakness of wrist and muscles of leg on same side; no absolute paralysis, but marked wrist-drop and great weakness of whole right side. No changes in sensation. Testicle objectively normal. Severe and constant headache. No irregularity of pupils. No stiffness of neck. No Kernig's sign. The patient gradually became weaker. On March 18th became very cyanotic and seemed unable to clear the lungs of the accumulated sputum. Signs in the chest were unchanged, except for the increase of loud bubbling rales.

19*th*. Blood cultures on various media entirely sterile. Patient died March 20th. Clinical diagnosis: bronchiectasis.

Autopsy. Both pleural cavities partly obliterated by old adhesions, mostly posterior. Lungs voluminous; surface roughened by fresh fibrinous exudate. On section the lung is foul smelling and studded throughout with irregular patches, which are prominent and rough on cut surface. These patches have usually a yellowish-white centre, surrounded by grayish-red areas. At one point in upper right lobe there is a small cavity with a necrotic lining and surrounded by an area of consolidation.

The bronchi are dilated and contain a considerable quantity of a thick, semi-fluid material. The trachea is much inflamed and the main bronchi have a circumference of 4 cm. below the first division. The bronchi in the greater part of the lung are not very greatly dilated, but have thickened walls. Toward the periphery of lungs, however, the smaller bronchi are dilated into spindle-shaped enlargements like beads on a necklace. These bronchi are dark red and apparently eroded. The bronchi to the upper lobes are less involved and cleaner than those below.

Microscopic examination shows numerous areas of bronchopneumonia tending to undergo necrosis in the centre. Necrosis frequently involves alveolar walls and adjacent bronchus. Considerable masses of bacteria and leukocytes may be seen in the central portions of these foci. Many bronchi are entirely eroded in places, the erosion extending to the alveoli, so that quite large cavities are formed, no trace of the bronchial wall remaining. In other parts the bronchi are only partly eroded and are very thin and dilated. The larger bronchi are not so extensively destroyed, showing merely desquamation of epithelium and infiltration of cells. The enormously enlarged bronchial lymph glands are œdematous, their sinuses filled with fluid, but not containing an excess of cells. The lymph cords are increased in bulk and the bloodvessels dilated. No areas of necrosis are present.

Permission for examination of the brain and spinal cord refused.

Cultures from contents of bronchi yielded bacillus influenzæ in great number with a few streptococci. From the washed and teased bronchial wall pure cultures of bacillus influenzæ were obtained, as also from the gangrenous areas. The lymph glands were sterile.

The bacterial masses above noted were seen in sections stained with methylene blue and eosin or with carbol thionin to consist almost entirely of tiny rods resembling the influenza bacillus. The infiltrating cells about the bronchial epithelium and extending down into the wall were frequently packed with these tiny rods. A few cocci and large bacilli were seen on the surface, but not deep in the tissues.

This case shows many of the features ascribed to acute influenza in the writings of Pfeiffer, Leichtenstern, Hitzig and others. The duration and slow development in this case would seem, however, to place it with the subacute or chronic cases. Here we have every reason to believe from cultural evidence during life and at postmortem that bacillus influenzæ was the principal if not the exclusive cause of the extensive pulmonary changes.

The case was in many ways clinically like a rapidly progressing phthisis, with the irregular fever and sweats, the loss of weight, and profuse expectoration and hemorrhage. Yet the daily examination of the sputum revealed no tubercle bacilli; and at autopsy the most careful search for tuberculous lesions, macroscopically and microscopically, was negative. The nervous features of the case strongly suggest the hemorrhagic encephalitis or "encephalitis grippalis" described by Leichtenstern, Königsdorf, Fürbringer, and others in association with influenzæ. In the absence of postmortem observations on the brain, however, the diagnosis can but be provisional. Prof. Osler suggested that brain abscess might account for these features of the case.

CASE II.—*Cough three years; worse in last year; large amount of foul sputum coughed up in paroxysms; emphysema; orthopnœa; no fever, chills, or sweats; no hemorrhages; bacillus influenzæ cultivated from sputum in practically pure culture; progressive weakening; steady increase in amount of expectoration; death; autopsy.*—B. G., male, white, aged forty-seven years, bookkeeper. Complains of "bronchitis." Admitted December 14, 1904. Died February 4, 1905.

Family History. Unimportant. No pulmonary disease.

Past History. Had scurvy in Africa with enlargement of liver; no pneumonia; no hæmoptysis; no night sweats; "severe catarrh" in 1893 and again in 1895; no other history suggesting influenza; habits good.

Present Illness. Began to cough three years ago. Worse last two years. Foul odor increasing and large quantities of sputum in paroxysms of coughing for a year past. Very severe paroxysms on rising, with evacuation of large amounts of sputum, greenish, purulent, rather thin and frothy. Paroxysms occurring several times a day and interfering with sleep at night. No hæmoptysis; no chills or sweats; no pain; has lost steadily in weight and strength in past year.

Physical Examination. Skin slightly cyanotic. Some clubbing of fingers. Chest rounded. Marked dyspnœa with activity of

accessory muscles of respiration. Percussion note hyperresonant throughout chest, except in back, where there is slight impairment over left base and right interscapular region. Breath sounds everywhere well heard, and accompanied by numerous moist and piping rales. No tubular or amphoric breathing. No cavity made out. No friction rub. Blood count showed 4,600,000; erythrocytes, 9600; leukocytes and hæmoglobin of 87 per cent.

Bacillus influenzæ in very large numbers were cultivated from the sputum, with a very few colonies of diplococcus pneumoniæ. The patient did not respond to treatment and favorable surroundings. His cough continued severe and the amount of sputum large. There was never any elastic tissue found and daily examinations for tubercle bacilli were negative. The patient gradually lost strength, and was more and more exhausted by the coughing paroxysms necessitated by the profuse expectoration. He died February 24, 1905, without any acute exacerbation of his trouble.

Autopsy. Both lungs bound by old adhesions posteriorly. Bronchi contain a frothy turbid fluid with sickening odor. Lung substance emphysematous. No areas of consolidation. Bronchi everywhere dark red, granular-looking, and extremely and almost uniformly dilated to periphery of lungs. No special saccular dilatations or cavities. No tubercles.

Microscopically, there was general emphysema. Epithelial lining of bronchi well preserved in the main and showing scattered areas of metaplasia into a stratified squamous type with many mitoses. Marked infiltration of mucosa with lymphoid cells. The epithelial layer invaded by polymorphonuclear leukocytes. Hyperplasia of peribronchial lymph follicles. Atrophy of muscular coat and increase of the peribronchial connective tissue, including in places atelectatic alveoli with cubical epithelium. No evidence of tuberculosis. Owing to the fact that the lungs were injected with formalin *in situ*, it was impossible to secure cultures from the tissues at autopsy.

Stained sections, as in the previous case, however, showed that the walls of the bronchi were infiltrated with leukocytes, containing great numbers of small bacilli like bacillus influenzæ. Very few pneumococci were found in the sections stained by Gram's method. Coarse non-Gram staining rods were found in the masses of secretion in the bronchial lumen. The intracellular organisms were all of the influenza type.

CASE III.—*Chronic cough for thirty years; acute influenza eleven years ago with increased paroxysmal cough and profuse expectoration; signs of cavity in both lungs; no hæmoptysis; no tubercle bacilli; no elastic tissue; influenza bacilli in pure culture from sputum; clubbed fingers; arthritis deformans.*—S. N., single, white, female, aged forty-nine years, seamstress. First admitted in June, 1899. Complained of cough and profuse expectoration.

Family History. Good; no tuberculosis.

Past History. Subject to colds and winter coughs from girlhood. Measles the only acute disease of childhood. "Rheumatism" for seven years past, with gradual stiffening of fingers, shoulders, knees, and hips. In 1894 patient had severe attack of la grippe, since which time cough has been worse and expectoration more profuse. No night sweats; no hæmoptysis.

Present Illness. Really dates years back, a history of chronic paroxysmal cough with exacerbations every winter. Sputum abundant, tenacious, greenish, foul.

Physical Examination. A thin, poorly-nourished woman. Well-marked arthritis deformans, involving both knees and elbows; slight involvement of shoulder-joints.

Lungs. Signs of diffuse bronchitis over both fronts. Distinct signs of cavity in both interscapular regions; signs varying before and after paroxysms of cough and expectoration. Numerous moist and sonorous rales throughout both backs and axillæ. No fever; no sweats. Daily examination of sputum negative for tubercle bacilli and elastic tissue.

Second Admission, January 18, 1905. Complaint "cough." Arthritis more marked, involving nearly all the joints to a greater or less extent. Well-marked spondylitis. Cavity signs (amphoric breathing, pectoriloquy, cracked-pot sound, etc.), definite in both interscapular regions where previously noted. Diffuse moist sonorous rales. Large amount of fetid sputum. No hæmoptysis. Bacillus influenzæ in enormous numbers cultivated from sputum. Also small numbers of pneumococci. Sputum constantly negative for tubercle bacilli and elastic tissue.

Cultures from joint fluids negative on all media.

CASE IV. *Twelve years' standing; acute onset; probable empyema; early cavity formation; thickened pleura; paroxysmal cough; profuse foul expectoration; severe recurrent hemorrhages for ten years past; marked scoliosis; extreme hypertrophic osteoarthropathy; exacerbations often attended by fever and sweats; no tubercle bacilli; no elastic tissue; influenza bacilli in sputum in great number and pure culture.* —H. H., white, male, aged twenty-nine years, baker.

Family History. Unimportant; no tuberculosis.

Past History. Always strong and well until onset of present illness. Never any pulmonary disease. Habits good. Hands and feet unusually large from babyhood. In December, 1893, patient had severe cold after exposure; pain in right side; chills and fever; cough; night sweats. Profuse greenish expectoration; never blood-tinged. In bed two months. After patient was up and about for two weeks he began to have severe paroxysms of cough in the morning, bringing up large amounts of yellowish-green sputum, rather sweetish and sickening, probably due to the discharge of an empyema through the lung. This paroxysmal cough persisted, and fifteen months later the patient had his first hemor-

rhage from the lungs and was admitted to the hospital in March, 1895.

On this admission the patient was emaciated and very pale. Great enlargement of bones of hands, feet, and forearms. Right shoulder lower than left. Right chest rather flat above. Left lung clear, except for scattered rales. Right side shows definite signs of much thickened pleura below fifth rib and suggestion of cavity at angle of scapula. Sputum abundant, bloody. No tubercle bacilli or elastic tissue.

In the ten years following the patient was readmitted sixteen times, on nearly every occasion for severe hemorrhage from the lungs. The deformity of chest and right lateral scoliosis became more marked. Signs of cavity quite definite in right scapular region. Densely thickened pleura on right side. Diffuse rales on left. Osteoarthropathy more marked. Patient had persistent paroxysmal cough and profuse expectoration, rather fluid and purulent, very foul at times. Fever and sweats in the periods of exacerbation. Between these more severe attacks patient able to work at his trade. Patient responded well to treatment (rest, good food, etc.), cough becoming much less frequent and sputum greatly diminished in amount. There was always at least one paroxysm in a day, however.

Last admission, February, 1905. Condition practically unchanged except for a dry pleurisy on left side. Influenza bacilli were obtained in absolutely pure culture from sputum and in enormous numbers at first. As patient's condition improved the number of bacteria greatly decreased, though there were always a few to be found. In the ten years' observation there was never any elastic tissue found nor any tubercle bacilli.

Intratracheal injections were particularly beneficial to this patient, causing rapid diminution in amount of sputum and great reduction in number of bacteria, a 10 per cent. suspension of iodoform in olive oil being used for this purpose.

CASE V.—*Cough for nine years; history of pneumonia; empyema; operation; contraction of pleura; gradual development of paroxysmal cough with profuse, very foul expectoration; right lateral scoliosis; exacerbations with fever and sweats; no hæmoptysis; no tubercle bacilli; pneumococcus from sputum with small proportion of influenza bacilli and a fetor-producing organism; operation for drainage of cavity.*—R. S., white, female, aged fifteen years, school-girl.

Family History. Unimportant.

Past History. No illness other than chickenpox till an attack of pneumonia (right side) at age of six years, followed after twelve weeks by empyema and operation. Since this time patient has had a chronic paroxysmal cough with profuse, very foul expectoration, worse in winter, often prostrating her. Never any hæmoptysis. On first admission, October, 1899, patient was found to have definite

signs of cavity in posterior and upper part of right lower lobe, there being a very noticeable intensification of the signs after the paroxysms of coughing. Left side clear, except for a few coarse rales. Some clubbing of fingers. Well-marked scoliosis to right. Sputum very profuse, very foul, of a greenish color. No tubercle bacilli or elastic tissue found. In the succeeding six years the patient was frequently readmitted with exacerbations of cough and expectoration, usually with some fever and occasional sweats; cavity signs in right lung persisting. She developed quite well, despite her infirmity. The cough and fetor were very distressing, however.

Last admission, January 19, 1905. Physical signs unchanged. Cough very distressing. Sputum from 200 to 400 c.c. in twenty-four hours. Influenza bacilli constantly present in sputum, but in small numbers, both on coverslips and in cultures. Pneumococcus in much greater preponderance. There was also isolated on one occasion a small micrococcus, which decolorized by Gram's method and grew only on blood-containing solid media, as will be noted later.

Patient was transferred to the surgical side and operation for drainage of the cavity in right lung was performed on May 23, 1905, by Dr. J. C. Bloodgood. Several ribs were resected and the lung exposed over the area of cavernous breathing. The lung was very atelectatic and dark red-like liver. Several small cavities were incised and drained. Patient is doing well two weeks after operation. Cultures from the contents of the cavities at operation yielded pneumococcus in great preponderance, with a few colonies of bacillus influenzæ. The surgical and pathological aspects of the case will be more fully reported by Dr. Bloodgood.

In addition to the above five definite cases of bronchiectasis there were two others of chronic bronchitis with probable dilatation of bronchi, from both of which influenza bacilli were obtained in absolutely pure culture. Both were men in middle life; had history of cough and profuse expectoration for years, worse in winter. Often fever and rarely night sweats. No hæmoptysis. In neither case could any tubercle bacilli be demonstrated by daily examinations.

A sixth case which has a bearing on the pathogenicity of bacillus influenzæ as found in the sputum in chronic cases will complete the list.

CASE VI. *Chronic bronchitis thirty years; slight hæmoptysis; localized pleurisy with encapsulated effusion; bacillus influenzæ in pure culture from sputum; two weeks later empyema with bacillus influenzæ in pure culture from pus; operation; recovery.*—A. B., male, white, aged sixty years, tailor.

Family History. Negative.

Past History. Always healthy, except for chronic cough, worse in winter. No history of any acute pulmonary disease preceding.

Present Illness. In the winter of 1904 the patient had a severe attack of bronchitis; spat a little blood; occasional fever and sweats.

Rather profuse expectoration. Never quite well since. Four weeks ago became much worse, pain in side, night sweats, hæmoptysis slight.

Physical Examination. Diffuse bronchitis. Slight movable dullness in lower left axilla and back; irregular fever and sweats. Small amount of fluid, straw-colored, obtained by aspiration. Cultures on ordinary media sterile. Septum abundant, tenacious, mucopurulent blood-streaked. Cultures from sputum on March 6th yielded bacillus influenzæ in absolutely pure culture. Patient discharged on request March 12th, not much better. Readmitted March 30th in practically same condition, though feebler and with more fever and drenching sweats. Moderate leukocytosis. Signs of pleural effusion. Small amounts of slightly bloody fluid aspirated on several occasions. This was, unfortunately, not saved for cultures. On April 11th a few cubic centimetres of pus obtained by aspiration and from this influenza bacilli grew in absolutely pure culture. Patient transferred to surgical side. Eighth rib resected and pleura found very thick. A considerable amount of grayish, odorless pus evacuated and cavity drained. Patient improved rapidly.

The bacillus of Pfeiffer seems to have been the direct cause of the empyema in this case, the sputum having yielded absolutely pure cultures of this organism for some time previous to the onset of the empyema, and the same organism, and it alone, being found in the pus from the pleura at operation by culture and cover-slips.

Cultural Methods. The sputum was collected in sterile cups and examined without delay. The washing method employed was substantially that described by Kitasato and by Lord. It was interesting to observe the striking difference in the bacteria found on coverslips made before and after washing. These were stained by Gram with bismarck brown counterstain in exactly the same way. While those made before washing showed usually a greater or less number of extracellular organisms (diplococci, streptococci, and other mouth bacteria), in addition to bacillus influenzæ, it was quite possible in most cases to wash the sputum so that the particles now showed only influenza bacilli, and these all intracellular. Pigeon-blood agar was used in most cases as a culture medium; in others sterile fetal blood from the placenta was added to the agar. On this latter medium it was interesting to note that involution forms appeared earlier and in much greater number than when pigeon, rabbit, or adult human blood was used. Some of these forms were so large and irregular as to give at first the impression of a mixed infection. Transfers to fresh media and control cultures of the same size and source on other blood media, however, produced in every case a pure typical growth of bacillus influenzæ. The gross appearance of the colonies on the fetal-blood agar was not different from that on the other tubes, being always the tiny, water-clear,

discrete colonies, exactly as described by Pfeiffer. The small Gram decolorizing micrococcus isolated from Case VI. has not been identified accurately. It grew only on solid media containing blood. It decreased rapidly in vitality, so that it was lost after the fifth or sixth transfer. Its most striking characteristic was the production on the blood plates of the intense sickening sweetish fetor common in bronchiectasis. In some respects it conforms to micrococcus catarrhalis though that organism grows on ordinary media, and I have been unable to find any reference to its production of fetor when grown upon blood agar. None of the other organisms grown from the sputum in these cases (bacillus influenzæ, streptococcus, or pneumococcus) produced any fetor in pure or mixed cultures on like media.

CONCLUSIONS. 1. The influenza bacillus is probably capable of producing extensive pathological changes in the lungs, leading at times to bronchiectasis (Cases I. and IV.). The organism may be a secondary invader or associated with other bacteria; the latter may be the preponderant factor. In our small series there was no clinical difference determinable between the pure influenza infections and the mixed or non-influenza cases.

2. The very close clinical resemblance of these cases of bronchiectasis to some cases of chronic tuberculosis with cavity formation is important, and may, in the absence of cultural investigations, lead to wrong diagnoses. Physical signs alone, therefore, cannot be relied upon for a positive diagnosis of tuberculosis.

3. Care should be taken to prevent the possible infection of others from these chronic cases having influenza bacilli in the sputum, as the organisms may not have lost their pathogenicity by a continued growth in these old infections.

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N. B.—Since the completion of this investigation, and while waiting for surgical interference in Case V., a second admirable and comprehensive paper has appeared by Dr. Lord (Boston Medical and Surgical Journal, May 11 and 18, 1905), which covers a much wider field and greater experience. With his conclusions my smaller series seem fully to agree.

REVIEWS.

INTERNATIONAL CLINICS. Edited by A. O. J. KELLY, A.M., M.D., Philadelphia, Pa. Vol. I., fifteenth series, pp. 312. Philadelphia and London: J. B. Lippincott Co., 1905.

THIS quarterly, containing many papers of interest to the advanced practitioner, comes as a welcome visitor. Opening with a practical presentation of cardiac asthma by Merklen, followed by a medical clinic by Robin, and an article on infected cholelithiasis by Chantford, we obtain a fair view of the modern French practice. Douglas offers a scientific study of the carbohydrates in Human Urine in Health and Disease, while the eye and hand in the Diagnosis of Heart Disease by Walsh, who is always suggestive, and Parkinson on the Early Diagnosis of Heart Disease in Childhood, and Manges on Aortic Stenosis and Adherent Pericardium would satisfy the internalist even if Kendict, in an excellent discussion on Intestinal Adhesions, were not added for good measure. In surgery Young presents a description of a valuable modification of Wolfe's or Krante's method in skin grafting. Dawbarn, in a characteristic, forcible, and practical article, writes on the Starvation of Malignant Growths by Depriving them of Blood Supply. Durante's new Operative Method for the Total Extirpation of the Larynx seems formidable, but if it obviates the danger of inhalation pneumonia, should be made use of. Hibbs formulates the treatment of knee-joint disease from the wealth of material at his disposal. Glenard's disease is likely to come more prominently, if it be needed, into the field of the physician through the very sensible article by Gallant.

The thoughtful general practitioner will find much that he recognizes in his daily work in the paper by Mills on Morphinomania, Cocamania, and General Narcomania and some of their legal consequences, for it is to him that such patients come at a time when unfortunate consequences are more easily prevented. Carr adds to the increasing list of instances of cerebellar tumor, and Bramwell gives an excellent presentation of two cases of ocular palsy probably caused by a lesion near the thermoidal fissure. The single paper on Anterior and Posterior Parietal Presentations of the Head in Slightly Flattened Pelves, by Jardine, completes the list of individual articles. The Progress of Medicine for 1904 occupies more than a hundred pages. Stevens presents Treatment, Edsall and Stanton

Medicine, and Bloodgood Surgery. We are pleased to see that treatment is not to be taken as synonymous with medicine, but that it covers a wider field than medicine and surgery combined. And to one who may practice in a narrower field than these two great divisions of the healing art we commend this section for the wisdom of the choice of a reviewer whose broad reading is reflected by the entire section. From this far too brief summary of the contents of this volume it is evident that the usefulness of this series is fully maintained and in source the papers are actually international.

R. W. W.

ORGANES GENITO-URINAIRES DE L'HOMME. Par HENRI HARTMAN. 8vo., in five parts, with 412 illustrations. Paris: G. Steinheil, editeur, 1904.

THIS volume thoroughly covers the subject and is up to date in the newer phases thereof, notably in the newer methods of urethroscopy and cystoscopy. The older parts of the subject are thoroughly gone into, notably the preparation and care of instruments, exploration of the urethra, prostate, and bladder. The chapter on injections, lavage, and instillation, while presenting nothing new, is in accord with our own authors, but the illustrations on urethroscopy and cystoscopy are profuse and easily understood. Part II. deals with the treatment of acute and chronic urethritis, foreign bodies in the urethra, and operations thereon, thoroughly and concisely. The operations on the urethra are considered from the point of operation, as perineal, rectal, and hypogastric, and here cystotomy and cystostomy are also considered. Lithotrity and Bottini's operation each have a separate chapter and are admirably treated. The treatment of ailments of the urethra, bladder, and prostate follow in the main the principles laid down therefor in this country. The therapeutics of ailments of the kidney, ureter, and pelvis contain minute direction for the physical examination of the kidney as also its functional examination, although the method advised for separate ureterization seems crude in the extreme and not equal to our own. Puncture, nephrectomy, nephrotomy, nephrolithiasis, nephrostomy, nephropexy, their treatment, operation, and accidents due to them, are thoroughly considered, as is the surgery of the ureter and pelvis. Part IV. considers the congenital organs, traumatisms, castration, epididymectomy, anastomiasis of the spermatic cord, here quoting Martin's case operated on at the University Hospital of this city. The treatment of hydrocele, varicocele, hæmatocele, and cysts of the cord and epididymis, as well as vaginitis, orchitis and elephantiasis, tuberculosis and syphilis of the testicle, follows the usual lines. The penile and pre-

putial conditions, the operation, together with the consideration of the vices of formation, occupy the rest of the volume, and aside from the illustrations, which are above the average, present nothing new and follow the usual lines. E. A. H.

DISEASES OF THE SKIN: THEIR DESCRIPTION, PATHOLOGY, DIAGNOSIS, AND TREATMENT, WITH SPECIAL REFERENCE TO THE SKIN ERUPTIONS OF CHILDREN, AND AN ANALYSIS OF FIFTEEN THOUSAND CASES OF SKIN DISEASE. By H. RADCLIFFE-CROCKER, M.D. (Lond.), F.R.C.P., Physician for Diseases of the Skin in University College Hospital; Honorary Member of the American Dermatological Society; Membre Correspondant Etranger de la Société Française de Dermatologie, etc. Third edition, revised and enlarged, with 4 plates and 112 illustrations. Philadelphia: P. Blakiston's Son & Co.

THIS new edition of Crocker's *Diseases of the Skin* is a ponderous volume of more than 1400 pages, half again as large as its predecessor. A long list of subjects not included in previous editions is considered, of which the most important are *x*-ray dermatitis, toxin serum eruptions, porokeratosis, granuloma pyogenicum, leukæmia, and pseudoleukæmia cutis, acne necrotisans, acne agminata, folliclis, folliculitis decalvans, and blastomycosis hominis. We note with hearty approval that the author, for the sake of uniformity of nomenclature, has changed the names of some of the diseases; and we sincerely hope other authors, influenced by this example, may likewise endeavor to bring some order out of the chaos of names in which dermatology has so long floundered.

Concerning the role of parasites in the causation of eczema the author's view is that "while a limited number of cases of local dermatitis indistinguishable from eczemas are parasitic, in most the dermatitis, however caused, only opens the door to the parasites whose presence keeps up local irritation, so that their destruction is an important step in the restoration of the skin *ad integrum*." He believes that toxins, having their origin most frequently in the alimentary canal, often cause eczema, although he admits the difficulty of definitely proving it. He accepts unreservedly the view that seborrhœic dermatitis, Unna's seborrhœic eczema, has a parasitic origin.

Psoriasis is believed to be due to an unknown microparasite which finds conditions suitable for its growth in the skins of certain individuals only. This parasite, while reaching the skin from without, probably reaches the circulation in a certain number of cases, causing the wide and rapid spread of the eruption which is not infrequently observed. In the treatment of this annoying and obsti-

nate affection the author accords a prominent place to salicin and its derivatives, remedies which he introduced to the notice of the profession in 1895. Salicin is preferable to the salicylates because it is much less apt to disturb the stomach. In doses of 15 to 20 grains three times a day it usually produces paling of the patches and a noticeable diminution of the scaling; and, unlike arsenic, it may be given in the acute stages of the disease without danger of increasing or aggravating the eruption.

Some 55 pages are devoted to the discussion of tuberculosis of the skin. This great increase in the space given to this most important subject, while due in large part to the numerous additions made to our knowledge in the past decade, is not wholly so, but results in part from the rearrangement of the subject-matter of the text by which certain diseases are now included among the tuberculous affections which formerly were given a place apart, as, for example, the scrofulodermata. The author seems only mildly enthusiastic concerning phototherapy in lupus vulgaris, and apparently is not yet quite ready to throw aside all the older methods of treating this distressing malady for the new one; he even believes there is still a place, although a limited and subordinate one, for the greatly discredited tuberculin.

Perhaps the most extensive alterations of and additions to the text are to be found in the sections on the vegetable parasitic diseases. These embody the very extensive increase in our knowledge of the ringworm fungi resulting from the work of Sabouraud and others during the ten years which have elapsed since the appearance of the second edition.

A short section is devoted to blastomycosis, an interesting and important parasitic affection of the skin whose clinical and pathological features have been made known to the profession chiefly through the work of American observers.

In conclusion, the same praiseworthy features which early won for this treatise an enviable place in dermatological literature, characterize this new edition, and we heartily recommend it to all those (and this should include every member of the profession) who are interested in this department of medicine. M. B. H.

MALADIES MICROBIENNES EN GENERAL. Par PAUL CARNOT. Nouveau Traité de Médecine et de Thérapeutique. Publié en fascicules, sus la direction de MM. P. BROUARDEL et A. GILBERT. Paris: T. B. Baillière et Fils, 1905.

THIS volume of two hundred and odd pages represents the first "fascicule" of the "Nouveau Traité de Médecine," edited by Brouardel and Gilbert. The book is to be published in pamphlet form.

Each pamphlet is to deal with one subject, and it is expected that the forty sections which go to form the system will appear in rapid succession until the work is completed. The present "fascicule" is evidently intended as an introduction or preface to the later monographs, for the subject of bacterial diseases is treated in the broadest possible manner and does not admit very well of detailed review. The work is not even divided into chapters. The subject receives very free treatment and forms itself into an expanded introduction to bacteriology, pathology, medicine, and therapeutics. The book is roughly divided into several parts: the comparative anatomy and physiology of the microbe and organic cell; the general etiology of infections; the general pathology of infections, including paragraphs on the virulence of bacteria, and the modern conceptions of immunity. The general pathological anatomy of infections, containing paragraphs on cellular degenerations and regenerations the general symptomatology of infections; the general diagnosis and prognosis of infections, and the general therapeusis of infections. None of the subjects are developed in detail, and even the question of immunity receives but short treatment. The results of modern investigations are very freely quoted and the authors' names mentioned, but there is unfortunately no bibliography. W. T. L.

LEHRBUCH DER KINDERKRANKHEITEN FUR AERZTE UND STUDIRENDE, VON DR. ADOLF BAGINSKY, a. o. Professor der Kinderheilkunde an der Universität Berlin, Director des Kaiser und Kaiserin-Friedrich-Kinderkrankenhauses. Achte völlig neu durchgearbeitele und verbesserte Auflage. Leipzig: Verlag von S. Hirzel, 1905.

THE demand for an eighth edition of Baginsky's *Lehrbuch* is a well-deserved compliment to the author and serves to emphasize the long-acknowledged excellence of this widely known classic. In the present edition the text has been thoroughly revised and some minor changes have been made in the systematic grouping of the sections on the diseases of the newborn, the gastrointestinal diseases, and the diseases of the special sense organs. Practically there was little to change in the already well-revised text of the preceding edition. As it now stands it must be acknowledged to be the most systematic, complete, and encyclopædic treatise on the diseases of children in any language, and as such it constitutes an indispensable adjunct to the library of every physician whose practice includes the care and treatment of children. T. S. W.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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1. **Double Heart Rate, with Remarks on the Analysis of Irregular Pulses.**—HOFFMANN (*Zeitschrift f. klin Med.*, 1904, Bd. liii. p. 206). 2. **The Paroxysmal Occurrence of Doubled Heart Rate.**—LOMMEL (*Deutsch. Arch. f. klin. Med.*, 1905, Bd. lxxxii. p. 495). 3. **Contribution to the Study of the Extrasystoles.**—GERHARDT (*Ibid.*, p. 509).—These three contributions present the most recent views on paroxysmal tachycardia. Hoffmann was able to obtain numerous pulse tracings at the beginning and termination of attacks, and makes the following interesting observation: With a pulse of a normal period of $\frac{4.6}{5}$ second, suddenly occurs a diminution to $\frac{2.3}{5}$ second, with each second beat much smaller in size than the preceding; after about seven such beats a still smaller one begins to be interpolated just in the middle of each beat, and this rapidly growing in size, the waves soon recur at regular intervals of $\frac{1.15}{5}$ second. These waves, it is true, are not all of one height, but the variations have no regular recurrence and the interpolated waves become indistinguishable from the original $\frac{4.6}{5}$ elevations. In other words, a pulse of 60 per minute rises suddenly to 120 and then rapidly to 240, so that the first rise is just twice and the second four times as rapid as the original normal pulse. The termination of an attack does not, as a rule, occur with such precision; the pulse becomes irregular and long and short periods vary for about three beats until the rate drops to normal. Even this irregularity, however, is remarkable, as the length of each individual beat represents a multiple of the length of the foregoing rapid beats. The venous pulse, too, during this decline is of great interest, as it shows distinctly that the auricle beats at the increased rate longer than the ventricle. Hoffmann goes over the literature carefully and selects many cases which he is sure must, if carefully observed, have shown the same condition as his own.

Such a proportionate increase and decrease in the rate of contraction of the cardiac muscle is not without analogy in physiological experiments. Kries has shown that by warming and cooling different parts of the heart the rate of contraction of the warmed part is always a mul-

tiple of 2 of the rate of the cooled part. Thus it may be 2, 4, 8, 16 times as rapid, but never 3, 5, or 7, etc. Gradual changes in the temperature were always associated with sudden changes in the rate, N beats going at once to N 2 beats and then to N 4 beats, etc. Engelman, also, has noticed in pieces of resected vein from the frog's heart that sudden single stimuli thrown in at the end of diastole will cause the muscle to make a number (from 2 or 3 to over 50) of rapidly succeeding contractions, the new rate being just double the original, while the return to the original rate is abrupt, not gradual, as in stimulation of the accelerator nerves. The origin of the cardiac impulse has been definitely placed in the vena cava, and it may be assumed that there is formed here a continuous and constant stimulation always ready to act but able to manifest itself only periodically on account of the refractory period of the muscular contraction, or that the stimulating substance is itself used up in discharging an impulse and must reaccumulate before being able to start a new contraction. As evidence favors the second view, changes in contraction rate should then depend upon positive or negative chronotropic[1] influences. The theory of a constant stimulus would involve the assumption of the existence of negative or positive changes in irritability. Either of these views explains satisfactorily enough the usual variations in heart rate, but there would appear to be no reason why they should act so as to cause abruptly a halving or doubling of the previous frequency. Analogy with physiological experiments suggests at once that there must be some change in irritability, while the stimulating centre continues to work rhythmically. To explain this consistently, Hoffmann suggests that higher up in the vein than where the normal impulse takes its start there may be muscle fibres in which is generated a rhythm faster than the normal heart rate. If these, for instance, develop 4-N impulses, and, as in Kries' experiment, the neighboring fibres are able to respond to only every second, these will take up a 2-N rhythm, and the point where the normal cardiac contraction has its origin, able to answer only every other impulse, will take up a 1-N rhythm. If, then, the conductibility, irritability, and contractibility of the heart and of the fibres at this point be increased, it may come to pass that instead of every other impulse, each individual impulse from the point above may take effect, resulting in a doubling of the heart's rate. Similar conditions occurring higher up would then allow a quadruple heart action. For the origin of such influences Hoffmann looks to the nervous system.

Lommel cites a case which corroborates Hoffmann's observations, and accepts his deductions almost completely. He lays special emphasis on the pulsus alternans character of the curves at the beginning and end of an attack. Hoffmann in 1900 had suggested the view that attacks of paroxysmal tachycardia depended upon a heaping of extrasystoles, the continuous irritation giving rise to premature systoles as soon as the bounds of the refractory period were reached. The perfectly regular rhythmical recurrence of the pulse waves, the absence of any compensatory pause over a long period of time, and the curious proportion of increase in rate lead both Hoffmann and Lommel to discard this view. Gerhardt, however, has something to say in its favor. His curves and interpretations cannot be reproduced, but he

[1] Influences involving the development of the stimulus.

believes he has shown that a doubling of the pulse rate, as well as a gradual increase in the size of the interpolated wave (as in Hoffmann's curves at the onset of an attack), may be produced by extrasystoles. He lays emphasis upon the tendency to the production of extrasystoles just before and after an attack of paroxysmal tachycardia.

SURGERY.

UNDER THE CHARGE OF

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Scopolamine-morphine as an Adjuvant in the Administration of General Anæsthesia.—SEELIG (*Annals of Surgery*, August, 1905) presents a series of 65 general anæsthesia administrations by this method to show that it merits recommendation and trial. The operations invaded the abdomen, cranium, neck, and rectum. In every instance the patient received a hypodermic injection of scopolamine hydrobromate gr. $\frac{1}{100}$, of morphine gr. $\frac{1}{8}$, one-half hour before the administration of the general anæsthetic, which was induced by the ethyl chloride-ether sequence. The Bennet inhaler, with a slight modification for the administration of the ethyl chloride, was employed for the general anæsthesia.

Scopolamine is an alkaloid from the hyoscyamus niger. Its sister alkaloids are hyoscine, daturine, duboisine, atropine, and hyoscyamine. By some authorities it is claimed that scopolamine is closely allied to hyoscine; by others that the two are identical, while Ladenburg claims that hyoscine and scopolamine are by no means identical.

Kochman states that there has never been a death from scopolamine. By the method of administration and dosage worked out by Korff, three doses are given, at three, one, and one-half hours before operation. Rational objections have been made to the fixed dosage. Three deaths have occurred.

Seelig is enthusiastic in its praise. Of his 65 cases, only 1 vomited or retched while on the operating table; 77 per cent. did not vomit at all. One-third of the cases that did vomit, vomited only once, and then only from 2 drachms to 1 ounce of clear mucus. Nausea was never pronounced, except in 2 cases, and vomiting never occurred earlier than two hours after the operation. Laryngeal sensibility was intact by that time and acted as a complete safeguard against tracheal inspiration, and the consequent danger of aspiration pneumonia. Besides decreasing the tendency to vomit, it lessened the quantity of the anæsthetic necessary, only 4 ounces per hour at operation being required.

By the administration of scopolamine-morphine the patients go under and come out from the influence of the general anæsthetic without passing through the usual stages of excitement. Salivation is almost invariably absent. Much less pain is experienced by the patient in the first twenty-four hours after operation. These advantages have been confirmed by Tuffier, Israel, Dork, and Robertson (Robertson used hyoscine instead of scopolamine). Morphine lessens the afferent nerve impulses, reaching the vasomotor centres, and thereby lessens shock (Crile). Scopolamine raises the blood pressure and thereby aids in preventing shock. The consensus of opinion is that many of the irregularities in the action of scopolamine are referable to impure preparations of the drug. Merck's scopolamine is the best preparation of the drug.

Scopolamine-morphine Anæsthesia.—Ries (*Annals of Surgery*, August, 1905) says that the greatest difficulty has been found in determining the best proportion of the two drugs to employ. Too much morphine will affect the heart dangerously, while too much scopolamine produces a state of excitement similar to that following atropine poisoning. The patients become restless and talk as if in a delirium. The pupils are widely dilated and rigid and the face is flushed. They demand water constantly and complain of dryness; but the dryness soon wears off, and neither in man nor in dogs have any cases become known where scopolamine alone has produced death.

Scopolamine-morphine is used for two purposes: First, with the intention of performing the whole operation under this anæsthesia alone; secondly, as a preliminary to anæsthesia by inhalation of chloroform or ether. If the case is a complete success the patient becomes sleepy after the first injection, is fast asleep after the second, and is unconscious and insensible to pain after the third. The operation may begin one-half hour after the last injection.

According to Ries' experience, the sleep may be so slight that the patient awakes when spoken to or moved. Some, if aroused, cannot answer intelligently, but mumble a few unconnected words. Others again, though they cannot be aroused, move when being handled, or even complain. It then becomes necessary to add some other anæsthetic to the scopolamine-morphine. Ries has, in a number of cases, added infiltration anæsthesia with a 0.06 per cent. salt solution without cocaine, to the complete exclusion of chloroform. When chloroform is given very little is required, and in many cases after the first few drops have been given for the first incision no more chloroform is necessary, so that operations lasting fifteen to thirty minutes can be finished with less than a teaspoonful of chloroform. Complete muscular relaxation does not, as a rule, result.

Ries has used scopolamine-morphine on 72 patients in 92 operations. Of the 72 patients, 3 died. In none could the scopolamine-morphine be accused. Of the deaths reported as due to it, only 1, in Ries' opinion, seemed to be due to the scopolamine-morphine. He has used hyoscine instead of the scopolamine, and with both had the same result.

Scopolamine, when dissolved in water, does not keep long, and it has been Ries' rule to have a new solution made every week. Even then it was noticed that the effect of the drug would decrease toward the end of the week, so that in the future he will have a new solution made every three days.

The Relationships between Colitis and Appendicitis from a Surgical Point of View.—Lockwood (*British Medical Journal*, March 4, 1905, p. 466) says that any form of colitis may be associated with appendicitis, and that it is of the very highest importance that we should try to learn their relations to each other. Without doubt appendicitis can cause colitis. In many operations for the removal of the appendix the end of the ileum, the cæcum, and the right colon are obviously inflamed. In addition there is tenderness along the course of the right colon and rigidity of the abdominal wall. Mucus or mucus tinged with blood in the stools has been associated, showing that inflammation on the outside of the colon must also have involved the mucous coat.

The relationship between mucous colitis and appendicitis is a mere question of extension by proximity, facilitated, perhaps, by identity of vascular supply. In cases of this kind the bowels soon recover when the appendicitis has ceased. The appendicitis is the dominant note. Colitis may be the prominent feature to such an extent that the appendicitis may be obscured and overlooked.

Two cases are referred to of appendicitis with left-sided pain, but with absence of pain and tenderness over the appendix. The cause of the left-sided pain was clearly colitis. In both the inflamed appendix was hidden behind the right colon, which might have afforded some sort of protection to it. The writer says that while he does not know that appendicitis can cause the mucomembranous form of colitis, he does know that mucomembranous colitis can cause appendicitis. In the last case referred to in his article, postmortem examination showed the whole depth of the mucosa ulcerated away, so that no trace of the tubular glands remained. The submucous, muscular, and peritoneal coats were much inflamed, with dilatation of their bloodvessels and lymphatics. There had been no reason to suspect that the appendix had suffered, but microscopically it was ulcerated exactly the same as the intestines.

On Ascending Currents in Mucous Canals and Gland Ducts and Their Influence on Infection.—Bond (*British Medical Journal*, July 29, 1905, p. 232) says that by some means or other and under certain conditions particles of an insoluble substance, such as indigo, inserted into the orifice of a mucous canal or duct are conveyed along the mucous channel in a reverse direction to that taken by the contents of the tube or by the secretion or excretion of the glands along the ducts. The conditions which seem to favor this passage are: First, some interference with the normal flow of the contents of the mucous tube or duct; some arrest or diversion of secretion, such as is produced by a fistulous opening, though it is by no means necessary that this should be complete.

That this movement is not due to ciliary action is shown by the fact that it occurred where cilia are absent. While irregular or reversed peristalsis might cause it, it is suggestive that the ascent of the indigo up the colon was not prevented but rather accelerated by the increased downward peristalsis produced by purgatives. Reversed muscular contractions are as yet, moreover, unknown or rather undemonstrated in the bile-ducts, urethra, and urinary tubes, Fallopian tubes, or in the ducts or glands, in all of which the reversed mucous currents were demonstrated. It is not due to a physical agency alone, such as capillary action. This is absent in the non-living tube.

The essential conditions seem to be a living tube, whose walls are

partially if not wholly in apposition and lined by a mucous secretion; while the reversal of this mucous current is favored by any condition (such as a fistula) which leads to arrest or diversion of the ordinary secretion of the viscus or gland.

The bearing of these remarks on the larger question of infection will be at once apparent. Bond is inclined to think that in some cases more attention should be paid to mucous channels as routes of infection rather than to the lymph or blood stream. Bearing in mind the proved transference of particles under certain conditions from duodenum to gall-bladder and from urethra to ureter, it is to be hoped that further light may thus be thrown on these calculous diseases.

THERAPEUTICS.

UNDER THE CHARGE OF

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ASSISTED BY

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The Treatment of Heart Affections.—DR. E. DE RENZI, from a prophylactic standpoint, advocates the administration of large doses of sodium salicylate (1½ to 2 drachms per day) in all cases of acute articular rheumatism and the proper treatment of all gouty tendencies and of obesity. In endocarditis with compensation, hygienic treatment is necessary, but digitalis is contraindicated. On the other hand, lack of compensation necessitates reliance upon this drug, the best preparation of which is, in the author's opinion, soluble digitoxin. Its action takes place quickly and it presents no disadvantages. Lesions of the heart muscle may be benefited by exercise, under the influence of which the myocardium becomes strengthened and hypertrophies. In this connection, Oertel's method increases the respiratory power, accelerates the elimination of toxins and facilitates the peripheral circulation. It is specially useful in fat patients and in functional heart lesions. Massage and Swedish movements lessen the peripheral resistance, dilate the capillaries, and militate against venous congestion. These are indicated in organic lesions.—*Berliner klinische Wochenschrift*, 1905, No. 11, p. 291.

The X-ray Treatment of Tineæ.—DR. DUPEYRAC reports the results of the treatment of a case of tinea tonsurans, a case of *trichophytosis barbæ*, and a case of favus of the scalp. These were well treated by an *x*-ray of slight penetration, and the surrounding parts were shielded by a protection of lead. In the first case epilation was complete on the twentieth day and a cure was achieved in three and one-half months; in the second case treatment was continued for a month, and two months

later the cure was complete. The third patient is still under treatment, but the ray does not seem to be in this instance superior to the forceps for purposes of epilation. The author considers that as he has employed them, the rays do not act as a parasiticide but only as a depilatory agent, thus making it possible to thoroughly clean the site of the lesion. There is difficulty in regulating the strength of the ray, so that it shall exert this depilatory action and yet not produce a dermatitis and complete alopecia, consequently he employs rays of slight penetration and of an amount corresponding to 5 units H.—*Marseille médicale*, 1905, No. 6, p. 173.

The Treatment of Gastric and Intestinal Hemorrhage.—Dr. Capitan considers calcium chloride the most useful drug in these conditions. In typhoid hemorrhage the baths are to be stopped, the patient is kept as quiet as possible; milk is replaced for three days by a fixed quantity of water. Two intestinal irrigations per day of a quart of water each at 118°, to which a drachm of calcium chloride has been added, are given. At the same time the patient is given 30 grains of this substance in aqueous solution by mouth. The irrigation must be given gently, slowly, and under low pressure. Four or five small doses of opium by mouth complete the treatment. Gastric hemorrhage is to be managed in practically the same manner. The intestinal irrigations may be supplemented by injections of serum and other means calculated to sustain the patient should be employed. The irrigations relieve the intestine of the extravasated blood, and, given at a temperature of 118°, they have both a constricting effect upon the bloodvessels and act as a nerve sedative. The calcium chloride increases the coagulability of the blood and enhances the hæmostatic effect of the hot water. This agent is not toxic, is not disturbing to the stomach, and is readily absorbed. Its action takes place in about fifteen minutes, but taken in larger doses than those above mentioned, it loses its power of increasing blood coagulability.—*La médecine moderne*, 1905, No. 10, p. 76.

Dechloridation in Epilepsy.—Dr. Arthur Morton reports encouraging results from the substitution of sodium bromide for sodium chloride in the food of epileptics. The sodium chloride was not wholly excluded from the diet, but enough of the bromide was added to the food so that each patient took about 15 grains per day. It is claimed that this salt is rapidly absorbed and becomes part of the body tissue when given in this manner and that only about half the usual quantity is necessary to produce the sedative effect. The author concludes that this method controls the convulsions and has little if any affect upon the nutrition. It is apt to cause constipation and does not furnish enough salt to satisfy the patient. It may be advantageously employed in intelligent patients, but is useless in middle-grade epileptics, as they have neither the desire nor the will to carry it out. A modified salt-poor diet, in which about equal parts of sodium chloride and bromide are used in the food, may be employed with advantage with idiotic and demented patients if their diet can be absolutely controlled. Bromism is comparatively rare.—*Boston Medical and Surgical Journal*, 1905, No. 24, p. 698.

Antistreptococcus Serum in Scarlatina.—Dr. L. Mendelsohn finds that the serum is very prone to produce eruptions in scarlet fever patients

and with the exanthem at times a slight degree of albuminuria appears. The injections are contraindicated when heart lesions are present. To produce its best effects, the serum should be given during the first week of the disease, and even then its influence upon the temperature and the eruption is slight. It does, however, favorably affect the angina, and severe throat complications are rare under its use; glandular enlargements seem to be less frequent in injected cases. The author gives the impression that he considers the employment of the serum of rather doubtful value.—*Deutsche medizinische Wochenschrift,* 1905, No. 12, p. 461.

The Treatment of Scarlatina.—Dr. E. Aussel discusses the treatment of scarlatina under three headings: (1) To combat the pyresis and sustain the organism. The temperature should be controlled by baths, beginning at 82°, and, if necessary, lowered to 68°. So low a temperature as the latter should never be employed unless a good reaction is obtained. The bath acts as a heart sedative and a nerve tonic as well as an antipyretic, and it is ill-advised to attempt great reduction of temperature by this means. Antipyretic drugs, save possibly cryogenine, should not be given. To sustain the system tonics, such as ammonium acetate, kola, or canella, are indicated. (2) To eliminate the toxins. Fluid diet and the baths assist diuresis, and this should be further augmented by the ingestion of plenty of fluid. Theobromine, 30 grains per day, is an excellent drug in this connection. Intestinal irrigations of saline solution, in the author's opinion, may produce albuminuria and œdema. The bowels should be kept freely open. The heart action should be watched, and if a tonic is necessary, sparteine sulphate, 0.15 to 0.3 grain, may be given hypodermically. Digitalis is toxic and caffeine excitant in children. (3) To stimulate the nervous system: alcohol in the form of champagne may be given in small doses and hypodermatic injections of camphor in oil may be employed. Heart failure is to be treated by baths at 90° to 95° and oxygen inhalations.—*Annales de la policlinique de Paris,* 1905, No. 4, p. 80.

Viferral, a New Hypnotic.—Drs. K. Witthauer and S. Gaertner report this combination of anhydrous chloral and pyridine. It is a white powder, slowly soluble in cold water, easily soluble in hot. It has a bitter taste, and is less toxic than chloral hydrate, does not disturb the digestion, nor depress the heart. In ordinary dose (15 grains), it causes a sound, restful sleep, usually without unpleasant after-effects. Rarely there may be slight headache. If given for considerable periods, a tolerance may become established.—*Thérapeutische Monatshefte,* 1905, No. 3, p. 143.

Electric and Hydrotherapeutic Treatment in Heart Affections.—Dr. G. Zimmermann finds that electric baths with the alternating or sinusoidal current may be employed advantageously in cardiac affections; the palpitation and dyspnœa are relieved, the appetite is increased, and the patient's sleep better; the pulse becomes slower, the œdema less, and the blood pressure higher. In cardiac hypertrophy, however, baths with the alternating current are contraindicated; here the iodides or the nitrites should be given to lessen the peripheral resistance. Baths with the alternating current act well in dilatation resulting from an atonic

myocardium, and in emphysema, but not unless the heart muscle is able to regenerate itself by compensatory hypertrophy. The author believes that these baths are chiefly useful in circulatory disturbances with low blood pressure, early failing compensation, moderate degrees of fatty degeneration, atonic conditions of the heart muscle and arterial system, and in pulmonary emphysema. While in sclerotic conditions the Nauheim treatment is more reliable.—*Münchener medizinische Wochenschrift*, 1905, No. 12, p. 549.

OBSTETRICS.

UNDER THE CHARGE OF

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Complications in Labor Caused by Anomalies of the Amnion.—BARDELEBEN (*Zeitschrift für Geburtshülfe u. Gynäkologie*, 1905, Band liv., Heft 2) draws attention to the complications which may result from *adhesions of the amnion to the fetus.*

In 2 cases of young primiparæ the uterus remained between pains so hard and firmly contracted as to occasion suspicion of uterine tetanus. On vaginal examination it was difficult to find the external os. The head was movable. The membrane seemed unusually hard and resisting, and when finally ruptured but very little amniotic fluid escaped. In 1 case spontaneous birth followed, and in the other delivery by forceps. Mothers and children recovered without interruption. In 1 case the placenta and membranes showed nothing abnormal, but the small size of the membranes; in the other the shape of the placenta was abnormal, the umbilical cord very slightly coiled, the chorion easily separated from the amnion, but firmly joined to the decidua; the amniotic cavity could contain only a fraction of the usual quantity of amniotic liquid.

The writer also reports 2 cases where the amnion had caused direct interference with the fetus. In 1 at the fifth month of pregnancy there was prolapse of the umbilical cord, the patient having had hydrorrhœa for two months before. The fetus developed outside the membranes for three months. The umbilical cord was disproportionately long and reached to the vulva. Occlusion of the cord caused fetal death and made it necessary to empty the uterus. The cervix was found unusually resisting; the uterus did not promptly contract, and profuse bleeding occurred.

In the second case the patient was thought to have placenta prævia; she was, however, in good condition and had not had sudden and severe hemorrhage. It was thought that the placenta had become entirely loosened and that the presenting part was acting as a tampon. On

examination the fetal head could be felt and in front of it over a great extent of the head a sponge-like mass. There was a cerebral hernia, and the midwife in examining had pierced the brain substance, which caused bleeding. A broad amniotic band extended from the sac of the cerebral hernia to the back of the fetus, to the placenta and also to the region of the left hand. This band ruptured when strong pains occurred and the child was delivered. The membranes were completely developed and easily separated from each other. In this case the amniotic band had prevented the descent of the child, while the abnormality in the cranium and the perforation of the brain had produced hemorrhage.

He further describes a case in which the fetus was outside the amnion, in which the membranes had ruptured and hemorrhage had occurred. On vaginal examination the cervix was found dilated and filled with fetal parts, which with difficulty could be distinguished. It was finally made out that there was a monstrosity with abnormalities in the membranes and a very large placenta. The umbilical cord was very short, but 14 cm.; as the fetus descended traction upon the cord loosened the placenta.

That such a complication can produce a fatal issue is illustrated by a case in which a multipara having a breech presentation and fully dilated cervix had labor cease after seven hours' duration. On examination an oblique transverse presentation was present, with an abnormality in the membranes, and also a rupture of the uterus. The left shoulder of the child had torn its way through the uterine wall; it was possible by very gentle traction to deliver a malformed fetus, placenta, and membranes. A thick amniotic band had caused the elbow to be strongly bent and held it in that condition. A similar condition was present in one of the knees. After delivery the rent was tamponed with gauze in the hope that the patient might recover. Fever and renewed bleeding occurred, and the patient perished on the ninth day from fatal hemorrhage. On autopsy a large hæmatoma was found on the left side extending to the kidney. In the wound in the perimetrium was a large-sized vessel, which was not completely thrombosed and from which came the fatal bleeding.

Disturbances of Digestion in the First Days of Life.—In the *Archiv für Gynakologie*, 1905, Band lxxv., Heft 2, KERMAUNER contributes an interesting paper upon the disturbances of digestion during the first days of the infant life.

The high mortality of the first year of life has been repeatedly demonstrated. From 22 to 25 per cent. is a moderate estimate of the number of infantile deaths during the first year. One-third of these arise from some disease in the digestive organs. From 70 to 80 per cent. of those infants dying during the first year had some disturbance of digestion. Furthermore, the greatest mortality among infants is in the first two months of life, diminishing steadily after that time. Kermauner believes that this arises through errors in the care of newborn children. In studying this subject, he examined the discharges from the intestines of the infants under hospital care and found abundant germs in the matter discharged from the intestines; this was independent of cases of infection arising among the mothers. In some cases the mother's milk contained staphylococci. He succeeded in reducing the morbidity and the mortality in the Heidelberg clinic by two procedures. The first was

a change in the manner of nursing these children, whereby an increased number of trained nurses was provided, and minute attention given to the atmosphere of the wards, to disinfection of diapers, the care of the umbilicus, the use of sterile powder, of sterile water for bathing, and also of soap. The closest attention was given to all the details of asepsis and sufficient nurses were employed to carry this out.

A second factor of improvement was the prevention of loss of heat on the part of the children; care was taken to keep the children properly clothed and to avoid exposure of the child at the time of bathing. The clothing of the children was not changed. A further precaution was the removal of the children from the bed of the mother; each child was given a small but separate crib, and contamination from one child to another was carefully avoided.

The results of this treatment were exceedingly good and demonstrated clearly the correctness of the reasoning which led to its institution.

Virulence of Streptococci Found in the Vagina of Pregnant Patients.—Walthard (*Zeitschrift für Geburtshülfe und Gynäkologie*, 1905, Band liv., Heft 2) has studied this subject by observing the agglutination of streptococci obtained from pregnant patients. Clinically speaking, he is unable to differentiate these germs from those of less degree of virulence; and his investigations show that streptococci remaining in the vagina of pregnant patients for a considerable time where the patient is not interfered with, so far as the patient is concerned, are practically non-virulent saprophytes.

Gangrene after Eclampsia.—In the *Monatsschrift für Geburtshülfe und Gynäkologie*, 1905, Band xxi., Heft 6, Gutbrod describes two cases of gangrene occurring after eclampsia.

The first was that of a primipara, aged twenty-two years, who had severe eclamptic convulsions and remained unconscious after the delivery of the child. She was treated by hot packs and perspired freely. In her unconscious condition she lay much of the time with the left hand under the back and the left foot crossed over the right. As she began to recover from eclampsia, gangrene of the left hand developed, while the skin over the sacrum against which the hand had pressed became also gangrenous. The same condition developed on the inner side of both knee-joints, on the under side of the left foot, and on the dorsal surface of the right foot. Forty-six days after the beginning of her illness the patient had improved considerably, but the tendons on the right foot had been exposed by slough, suppuration burrowed into the foot, and it was finally necessary to amputate it.

The second case was that of a multipara who had eclampsia with spontaneous expulsion of the child. After labor the eclampsia persisted; the patient had profuse bronchial catarrh, with threatened œdema of the lungs, which gradually subsided. She remained unconscious for some time after delivery, and on the fifth day after confinement the great toe of the left foot began to turn black. The patient had fever, and the entire foot became deeply red. Her convalescence was very slow, as pus formed which required incisions in several parts of the body, and the cornea became clouded. Over four months was consumed in the patient's convalescence. She finally made a good recovery.

GYNECOLOGY.

UNDER THE CHARGE OF

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Periappendicitis Decidualis under This Term.—HIRSCHBERG (*Zentralblatt f. Gynäkologie*, 1905, No. 28) describes a peculiar condition seen in an appendix which was found to be adherent to a tubal mole. Microscopic examination showed that in addition to the hypertrophy of the peritoneal endothelium due to the irritation of the effused blood, there was an actual growth of decidual cells, while in the serosa itself were isolated groups of such cells. Schmorl has shown that even in normal pregnancy the cells of the peritoneum may undergo a similar change, as shown by the "decidual reaction," or the homogeneous staining of the protoplasm. The same writer states that while such cells are found in the non-adherent omentum, they may be absent when it is attached to the uterus.

Report on 1000 Laparotomies.—VON PODHARITZKY (*Archiv f. Gynäkologie*, Band lxxiv., Heft 3) reports a series of 1000 abdominal sections performed by Fenomenoff in the course of seventeen years. No antiseptic solutions were used during the operations, the hands and field of operation being cleansed according to Mikulicz's method with tincture of gum soap, alcohol, and tincture of iodine. The suture material was silk boiled in sublimate solution. There were 500 ovariotomies and 280 supravaginal amputations, 60 salpingo-oöphorectomies, and 58 cases of ectopic gestation; in addition to this, Cæsarean section was performed six times and the abdomen was opened seven times for rupture of the uterus. The combined mortality was only 5.1 per cent.

Calcification of the Fimbriæ of the Tubes.—WAGNER (*Archiv f. Gynäkologie*, Band lxxiv., Heft 3) describes the following condition found at autopsy: The fimbriæ of the tubes were deeply congested and yellowish-white in color and were covered with small granules. Under the microscope the latter were found to be small calcareous nodules in the tissue of the fimbriæ, which were swollen and their vessels congested. In a few places the intima was thickened and calcareous. No nodules were present in the connective-tissue spaces. The writer refers to a similar case described by Schmitt, in which the nodules were referred to persistent circulatory disturbance in consequence of cardiac insufficiency.

Operation for Prolapse of the Rectum.—WENZEL (*Deutsche Zeitschrift f. Chirurgie*, Band lxxvi., Heft 1) refers to the older methods of treating rectal prolapse with the cautery, ligature, and the more modern procedures of resection and suspension. He believes that in many cases the cause is due to the unusual depth of Douglas' pouch, and that the object

of the operation must be to fold in the peritoneal pouch, as well as to suture the relaxed muscles of the pelvic floor and external sphincter. In one instance after opening the abdomen he first suspended the retroflexed uterus, then dissected off the peritoneum from the uterus, vagina, and rectum, and sutured the rectum to the posterior surface of the uterus and vagina, fixing the sigmoid lateral and in front to the abdominal wall in order to prevent a kink in the gut. The operation was successful, but the patient was obstinately constipated and the prolapse gradually recurred. The writer thinks that the latter might have been avoided by simultaneously contracting the sphincter.

Resection of the Bladder for Cancer.—GOLDENBERG (*Beiträge zur klin. Chirurgie*, Band xliv., Heft 3), in reporting a case, refers to the 57 other reported cases, in which the greatest trouble was experienced in managing the ureters. Implantation in the rectum is not admissible, on account of the danger of ascending infection; on the other hand, the trigone is so often diseased that they cannot be sutured here. The writer has experimented with dogs with the view of utilizing the lower portion of the ileum for this purpose, a coil of gut being sutured to the abdominal wall and opened. When the ureters have become firmly attached to the gut the intestinal fistula can be closed.

Intermenstrual Pains.—VAN DE VELDE (*Zentralblatt f. Gynäkologie*, 1905, No. 30) believes that the periodical variations in the menstrual blood pressure are due to some chemical irritation proceeding from the ovaries. From observation of various blood curves and experiments with ovarian tablets, he infers that the menstrual flow is due to a falling of the pressure or general diminution of the vascular tone. Patients with a high blood pressure are apt to have so-called intermenstrual pains, which he explains as due to passive hyperæmia of the uterus. These are noticeably relieved by the administration of ovarian extract, which modify metabolism so that the wave sinks.

Double Ovariotomy during Pregnancy.—HEINSINS (*Zentralblatt f. Gynäkologie*, 1905, No. 32) reports the case of a five months' pregnant woman with severe abdominal pain and ascites. A movable pedunculated tumor the size of two fists was found in the right iliac region. On opening the abdomen this proved to be an ovarian dermoid with a complete twist of the pedicle and firm intestinal adhesions. A second dermoid was removed on the left side, thus being a torsion of 180 degrees. Recovery without interruption of the pregnancy.

Sexual Irritation in Early Tuberculosis.—LANDRET (*Zentralblatt f. Gynäkologie*, 1905, No. 32) calls attention to the fact that in the early stage of tuberculosis there may be increased sexual desire and capacity for procreation. He attributes this to the presence of the tuberculous toxin in the system, and thinks that it may prove valuable as an early diagnostic symptom.

Free Bodies in the Tubes.—FEDOROW (*Ann. de gyn. et d'obstétrique; Zentralblatt f. Gynäkologie*, 1905, No. 32) found in a sactosalpinx fifteen

free nodules varying in size from a pea to a bean, of different shapes, round, oval, and polygonal. Over the surface of the latter were scattered small villi. The bodies were soft, encapsulated, and microscopically presented the appearance of adenoma, with small cavities containing papillomatous masses. When the fibrous capsule was thick the interior of the body showed marked degeneration, containing only empty, atrophied bloodvessels, and necrotic epithelium. The influence was that there were papillomata that had become detached from the living membrane of the tube.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

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Treatment of Severe Blepharospasm by Artificial Anastomosis of the Facial with the Spinal Accessory Nerve.—ABADIE et CUNEO, Paris (*Arch. d'Oph.*, April, 1905), have successfully practised section of the facial nerve, followed by immediate anastomosis with the eleventh pair in a severe case of this affection, which had been rebellious to both medical and surgical attempts at cure. Section of the facial nerve is followed by a temporary paralysis; the latter soon recovers.

Aural Nystagmus.—BARANY (address before the Vienna Ophthalmological Society, *Centralblatt f. Augenh.*, August, 1905) was able to excite nystagmus by syringing the external auditory passages with water. He experimented upon 100 individuals, including some whose hearing was normal, some with aural disease, and some were deaf mutes. His results were as follows:

1. If the temperature of the water injected was below that of the body an almost purely rotatory nystagmus was excited directed to the opposite side of the body; the phenomenon was most marked as the patient looked toward the side opposite that of the ear which was being treated. If the injected water was above the body temperature an almost purely rotatory nystagmus was also excited, but it was directed toward the side of the ear which was under treatment and most marked as the patient looked in that direction. These phenomena only occurred with the head erect.

When the patient's head was inclined toward the side not under treatment there occurred in the first case (water below body temperature) a horizontal nystagmus directed toward the side under treatment; in the second case (water above body temperature) toward the side not

treated; this gave way to the above-mentioned rotatory nystagmus only as the head was erected or inclined toward the side under treatment.

2. If the injected water be of the temperature of the body no nystagmus occurs.

3. The nystagmus mentioned under 1 may be excited from either ear, the reflex paths being intact; nevertheless longer time and greater temperature differences are required where the drum membrane is present. Where the wall of the labyrinth is exposed the nystagmus occurs with very small temperature differences and at the expiration of five to ten seconds.

4. If the reflex path has been interrupted nystagmus cannot be excited from the affected ear, but it may be produced as above described by acting upon the other ear if it is sound.

5. The nystagmus is usually accompanied by vertigo and disturbance of equilibrium, occasionally also by nausea, faintness, and vomiting. All of these symptoms may be absent.

Where the disturbance of equilibrium is marked the subjects fall to the ground; the falling occurs in a certain definite direction; it agrees in general with that observed in disease of the vestibular apparatus.

As to the cause of these phenomena, the speaker regarded it as probable that the warming and cooling of the contents of the labyrinth acted as an irritant upon the terminals of the nerves. It has been noted that temperature stimuli may excite attacks in case of Meniere's syndrome.

Is Trachoma Contagious?—This question is hardly worth while discussing. Still, as there appear to be a few ophthalmologists who have some doubt upon the subject, it may be worth while to put on record an instance as typical as the one reported by POULARD (*Arch. d'ophthal.*, August, 1905), where all the members of a family and others who were brought into intimate contact with them became successively affected with the one disease.

The other inhabitants of the tenement in which the family resided were convinced of the danger of contagion and finally succeeded in having the family expelled, because, following their expression, "they were giving sore eyes to the whole house."

Insufficiency of Convergence.—LANDOLT (*Arch. d'ophthal.*, July, 1905) reports a case of this condition which is remarkable from the completeness of the result obtained by an operative procedure, of a kind which on first thought would hardly seem capable of leading to that result.

A girl, aged thirteen years, suffered from a rebellious asthenopia, which had long disabled her from doing fine work in a sewing establishment where she was employed. The left eye showed myopic astigmatism 0.50 D.; the right was emmetropic. The vision in each eye equalled 1.25. The accommodation was normal in both eyes. The cause of the complaints was found in defective convergence, which, indeed, was highly faulty. The maximum of this function was only 2.5 metre angles. The minimum of convergence, which is normally negative, was positive and corresponded to +1.75 metre angles, so that the patient could not get the visual lines parallel, the punctum remotum being situated at —0.507 in front of her. There was thus

present besides the insufficiency of convergence, that is to say, relative divergent strabismus, a convergent strabismus of 5.8. There existed in fact crossed diplopia inside of 40 cm. and homonymous diplopia beyond 50.

The history explains this strange condition. The child previously had had a tenotomy of the right externus performed, no doubt with a view of correcting the insufficiency of convergence, under the fatal error that weakening the external recti should strengthen the power of the interni. The operation, which was quite a moderate one, had for its effect to narrow the field of excursion of that eye. While the excursion outward of the left eye equalled 58 degrees, it was only 40 degrees in the right eye. The movements inward were 44 degrees right and 42 degrees for the left eye. This apparent gain of 2 degrees cannot be certainly set down as due to the operation, because the adductive power before the operation is not known. If it be admitted that this was equal in the two eyes, this increase of 2 degrees would have been paid for by a sacrifice of 18 degrees of adductive power.

In any case the tenotomy of the right externus had the effect of transforming the facultative divergence into a convergent strabismus with homonymous diplopia, while leaving a considerable insufficiency of convergence.

The condition was very embarrassing. Theoretically, it seemed necessary to detach the muscle unfortunately set back and reattach it to its normal insertion and to advance one of the internal recti to remedy the insufficiency of convergence. It was finally decided to attempt the re-establishment of the power of positive convergence by advancing one of the interni. The left was selected. Improvement was rapid, the amplitude of convergence increased in both directions, the punctum remotum receding while the punctum proximum approached the eyes. Ten days after the operation the minimum of the convergence was 0.75, the maximum 9 metre angles at the end of two months, the minimum had become negative, —0.50, while the maximum was 10 metre angles, that is to say, quite normal. A month later the conditions were still more favorable, having indeed surpassed the normal at both extremities, extending from —1.75 to 12 metre angles.

The advancement had thus resulted in a gain of 13 metre angles, divided between 9.5 in the direction of the punctum proximum and 3.5 toward that of the punctum remotum.

It is very remarkable that the advancement of the internus, while so greatly augmenting the power of convergence, did not also increase the convergent strabismus. On the contrary, this strabismus slowly disappeared and even gave place to a marked facultative divergence. The result obtained in this case, if we base our views upon the old but erroneous theory of the influence of muscle operations upon the movements of the eyes, is incomprehensible. It is explicable upon the view that tenotomy, that is to say, setting back of a rectus, causes the eye to advance outward, so to speak, from its muscular funnel and tend to lose excursive power, not only in the direction of the muscle which has been set back, but even on the opposite side. By advancement, on the contrary, we sink the eye more deeply into this funnel and give the muscles greater influence upon the rotation of the globe leading to increased power of all the muscles affecting the rotation of the eyeballs.

[This case is certainly remarkable. Were it not for the eminence and acknowledged skill of the reporter, one might be inclined to suspect that a neurotic element may have been present and that hysteria and suggestion entered into the symptoms and the cure.—ED.]

OTOLOGY.

UNDER THE CHARGE OF

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Regional Anæsthesia of the External Auditory Canal.—P. LAVAL (*Archiv f. Ohrenheilkunde*, Band lxiv., Heft 2 and 3), on the recently published experiments of von Eiken on local anæsthesia of the external auditory canal, with secondary anæsthetic effect upon the middle ear permitting removal of the ossicles, has led the writer to publish his own experiments, made by a similar method, independent of those of von Eiken's in the past summer in this field of work and since successively repeated.

Since the previously proposed methods of local anæsthesia had proved inadequate for special reasons, the Schleich infiltration anæsthesia limiting the space anæsthetized by wheal formation, the ethyl chloride anæsthesia being hardly applicable on account of the severe after-pains, and the higher per cent. solutions of cocaine having proved ineffectual, the writer adopted the method of regional anæsthesia by injections along the course of the nerves supplying the external auditory canal, the procedure being, therefore, an application of Oberst's original method for anæsthetizing fingers and toes, developed in later years by Braun.

The field of operation in the external canal and middle ear is supplied by three nerve trunks with sensory fibres, whose anatomical relationships were determined by successive comparative dissections in three cadavers.

Anteriorly the nervous meatus accustici externi enters a branch of the auriculotemporalis with two or three fibres to the external auditory canal, then it breaks through the fibrocartilaginous part close to the lateral part of the osseous canal, in order to ramify inward and outward in the fibrocartilaginous canal. Its outer limits of ramification lie close behind the crest of the tragus, inward it takes part in the innervation of the drum membrane. Similarly situated is the ramus auricularis vagi, which passes through the canaliculus mastoideus laterally from the os tympanicum to the fibromembranous canal and divides into two branches. It supplies the posterior wall, the greater part of the drum membrane, and a zone about 0.5 cm. broad and applies itself half-moon shaped in the *cavum conchæ* to the entrance of the external auditory canal. The

third nerve is a branch of the nervous auricularis magnus, which ascends with greater regularity from below to the sternocleidomastoid muscle, running subcutaneously in the axis of the antitragus to the posterior surface of the concha, and there dividing into several branches, distributing itself in the region of the cauda helicis to the lateral surface of the auricle. It supplies a part of the concha, lying below a line drawn horizontally through the tragus, as well as the lobulus, with sensory fibres, but on the posterior surface the line lies about 1 cm. lower.

In the greater number of operations, in the region of the external auditory canal, only the first two named nerves come into consideration, though there were cases in which a painless operation could be assured only by anæsthetizing the third nerve also.

In seeking the anterior branches it is possible to injure the temporal artery, the auriculotemporalis nerve, the capsule of the maxillary joint, and the parotid gland. However, if the mouth of the patient is opened as widely as possible the first two named organs sink deeper inward, together with their surrounding fatty tissue, as is easily demonstrable on the cadaver, and as can be tacitly appreciated by pressure in the groove in front of the tragus in the living subject, the folds of the capsule of the joint being tightly stretched and the parotid gland with the ascending maxillary branch being thrown forward. The facial nerve need not be considered, since it runs at the lower end of the external auditory canal, and the canal itself is avoided in the anterior as well as in the posterior injection by pulling the auricle upward and backward.

After disinfection and anæsthetizing the outer skin with a spray of ethyl chloride, the needle of a Pravaz syringe is inserted about 0.5 cm. in front of the tragus at the height of and in the direction of the floor of the external auditory canal perpendicularly to a depth of 1.5 cm., the injection fluid being made to flow during this process by constant pressure, the whole amount injected being 0.5 cm. The mouth should be widely opened and the auricle fixed by a gentle pull on the lobule. Where it is possible to open the mouth only partially rotation of the jaw toward the opposite articulation in the vertical axis will produce a compensating effect. The branch of the vagus is reached at the height of the canal by gently pulling the concha outward and forward and going in about 1 cm. in depth perpendicularly, close behind the fold of the auricle, between the cartilage and the bone of the mastoid process. From the same puncture opening, when necessary, the branch of the auriculus magnus can easily be reached, the needle, which is not completely removed from the puncture channel after injection, being pushed close under the skin about 1.5 cm. parallel to the posterior auricular fold forward and downward, and the final injection made into the subcutaneous adipose tissue close behind the lobule.

For all three injections 0.5 cm. of the solution is sufficient. Laval found that this quantity of fluid saturated the loose adipose and connective tissue through an area equivalent in extent to the size of a large cherry, and by bilateral injection of two cadavers with a weak watery solution of methylene blue, demonstrated that the nerve *trunk* was washed by the fluid to a great extent. In very fat individuals, in order to make sure, the dose should be increased to 0.75 cm.

The fluid used for injection in the clinical experiments was a solution of Braun's cocaine suprarenal tablets dissolved in 1 cm. of distilled

water. Each tablet contained cocaine muriate, 0.01; suprarenal borate, 0.00013, and sodium chlorate, 0.009, the result being a 1 per cent. solution of cocaine, the addition of 1 cm. of salt solution affording the equivalent of 0.5 per cent. solution of cocaine in physiological salt solution. In the first experiments a weaker solution only was used, but in the case of a strong thirty-five-year-old man, it did not diminish the sensibility, and the writer has since then favored the 1 per cent. solution for men, and for women and children the weaker 0.5 per cent. solution. The anæsthesia begins in about five minutes and lasts usually fifteen to twenty minutes; in one case, a girl, aged eighteen years, it lasted over three-quarters of an hour. In all cases the injection, as well as the operation of puncture, was painless and no evidences of cocaine intoxication were observed. As to after-pain, aside from slight sensitiveness of the place of injection for some time after the operation, there was no reported complaint on the part of any of the patients.

In the summer of 1902, 10 or 11 cases were operated upon under anæsthesia and 4 more in the following winter. The majority of the operations consisted in incision and curetting of furuncles in the canal. Of these, 1 is worthy of notice, a case in which a furuncle became an extensive abscess, completely closing the entrance to the canal and extending about 1 cm. downward in the cavum conchæ. By the third injection at the posterior point of puncture, the writer succeeded in anæsthetizing the lower half of the auricle, made a long incision, and curetted without causing pain. In 2 cases there were polypoid granulations about the size of peas, one from the edge of a deep ulcer, the other, with total defect of the drum membrane, resting on the posterior inferior part of the carious annulus tympanicus. A case of carcinoma of the canal in a man, aged about forty years, was of especial interest. The suspicious tumor, half the size of a pea, rested posteriorly and inferiorly on the lateral border of the middle third of the external auditory canal. The tumor was completely removed by an oval incision, including a strip of normal skin, then elevated from the bone and removed with forceps without causing pain. The result of the histological examination gave indications for radical operation. Close to the tumor there was total defect of the drum membrane, the malleus lying free, and cholesteatoma of the mastoid process, the middle ear being epidermatized. The tactile examination of the usually sensitive medial wall and attic of the middle ear by means of a probe, and even a relatively strong pull on the malleus, were unaccompanied by pain. It is to be assumed that, during the ten years' suppuration in this latter case, the nerves of the plexus tympanicus had degenerated and had been vicariously replaced by fibres of the nerves of the canal.

The result attained in these cases has led the writer to use local anæsthesia of the external auditory canal in incising the drum and in extraction of the malleus and incus. In the latter operation the anæsthesia failed of full effect, but a diminution of sensibility was observed in incising the drumhead, and once, according to the statement of the patient, it was completely painless. The mucous membrane layer of the drumhead obtains its sensory fibres from the plexus tympanicus, and these are not reached by the injection.

The results show the usefulness of local anæsthesia in the important field of the surgery of the external auditory canal. Furunculosis, which well demands the use of the method in the paramount number of cases,

can be painlessly treated, and, on that account, more thoroughly than without local anæsthesia. Small tumors, even of malignant nature, as one case showed, can be easily and completely removed.

If the inflammatory and painful area extends, as in one case, into the lower part of the cavum conchæ, the author recommends, in addition to the typical injection before and behind the canal, the one below the concha. This latter injection alone being sufficient to permit operations upon the lobule without sensation of pain.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Racial Differences in Strains of Typhoid Bacillus, and the Inhibiting Substances in Serum and Their Importance for the Gruber-Widal Reaction.—FALTA and NOEGGERATH (*Deut. Arch. f. klin. Med.*, 1905, Band lxxxiii. p. 150), in their work upon the agglutination of typhoid bacilli, bring out certain important points bearing upon the practical application of the Grüber-Widal reaction. From their investigations they conclude that the delayed agglutination or complete failure of agglutinations of the serum of certain cases of typhoid fever is to be explained in the first place by the fact that the strains of typhoid bacilli used for the reaction are only slightly agglutinable for that particular serum. This error may be rectified by using many types of typhoid bacilli. Substances which inhibit agglutination are frequently found in fresh human and animal typhoid serum. Often they may be demonstrable for the first time toward the end of an attack, though sometimes they are entirely absent. These substances are not identical with the proagglutinoids of Eisenberg and Volk, but are more nearly allied to some of the by-products of the thermolabile agglutinins of Joos. When they are present in large amounts they may give the false impression of a negative reaction.

Contribution to Our Knowledge of the Anticoagulating Substances of the Blood and Tissues.—CONRADI has demonstrated that the juice expressed from the organs and tissues of animals accelerates in an extraordinary way the coagulation of blood, while autolysis of the organs gives rise to substances which retard or suspend coagulation.

PUGLIESE (*Jour. de phys. et de path. gen.*, 1905, tome vii. p. 437) has discovered that from saline extracts which favor coagulation an antithrombine can also be obtained. Of more importance is the fact, which the author believes he has demonstrated for the first time, that an anticoagulating substance is present in the blood itself, or at least in the blood of dogs and certain birds. The blood of birds gives an extract which is more active than that of dogs, but contains less antithrombine than peptone blood. Pugliese concludes that in the blood there are coagulating and anticoagulating substances, which, as has been suggested by Ellinger and Spiro, may circulate in equilibrium. The first may be produced directly in the blood by the destruction of the formed elements, while the second or anticoagulating substances are thrown into the circulation by the organs and tissues, especially the liver. These anticoagulating substances pass through the circulatory system and neutralize the thrombine or arrest the transformation of zymogen into fibrin ferment. When the blood is drawn from the vessels it no longer receives anticoagulating substances from the tissues, and therefore only remains liquid for a varying time, depending upon the amount of antithrombine which it contains.

Enzymes and Antienzymes of Inflammatory Exudates.—OPIE (*Journal of Experimental Medicine*, 1905, vol. vii. p. 316) has studied, by means of autolysis, the proteolytic ferments in sterile exudates obtained by injections of aleuronat into the pleural cavity of dogs. The cells of the purulent exudate, as it is obtained, contain a ferment twenty-four to forty-eight hours after injection, though the ferment action is more marked three or four days after injection of the aleuronat. The amount of proteolytic digestion was determined by estimating the nitrogen of the uncoagulable proteid of the autolysed exudates. The cells of the exudates washed free of serum showed much greater autolysis than the original fluid, and further experiments showed that this fact was due to the presence of an antienzymotic action of the serum. This power of inhibiting the action of the proteolytic ferments contained in the leukocytes is also present in the blood serum, whence it probably passes into the inflammatory exudate. The antibody contained in the serum is destroyed by a temperature of 75° C. The proteolytic ferments of the leukocytes act both in an acid and in an alkaline medium, but are most efficient in the latter. The antienzymotic action of the serum is favored by an alkaline reaction, but is completely lost in an acid medium.

Experimental Studies in Yellow Fever and Malaria.—*Bulletin* No 14, Yellow Fever Institute, forms the report of Working Party No. 2, under the direction of Rosenau, Parker, Francis, and Beyer. The work of this party was carried on at Vera Cruz, Mexico, during the late summer and autumn of 1903. The party directed its work, especially toward investigating the cause of yellow fever. They first studied the bodies described by Parker, Beyer, and Pothin as myxococcidium stegomyiæ, which were considered to be animal parasites in mosquitoes. The party found these bodies, however, in many normal mosquitoes, and from their work conclude that they are yeast cells. They were unable to find in the blood of yellow fever patients, by the use of various stains, anything that might bear a casual relation to the disease, although they

consider it proved that the infection of yellow fever is in the blood serum early in the disease.

They found that after blood serum drawn from a yellow fever patient had been passed through a Pasteur-Chamberland B filter it was capable of producing yellow fever when injected intravenously into non-immunes. The blood serum used was obtained from an individual in whom yellow fever had been produced experimentally by the bite of a mosquito. After filtration an equal amount of normal salt solution was added to the serum; 5 c.c. of this mixture was injected into two non-immunes, and 3.5 c.c. of it was injected into a third. All developed typical attacks of yellow fever, although carefully guarded from infection; all recovered. Because the virus can be passed through this filter is no reason, according to the authors, for assuming that it is necessarily ultramicroscopic, for further experiments showed that carbon particles visible with Zeiss lenses would also pass through the same filter.

The authors' studies show that the period of incubation of yellow fever is from three to six days, usually the former, in authentic cases produced by bites of mosquitoes. When the disease is produced by the inoculation of serum the incubation period is less regular. Their work confirms that of Reed and his party in that they found that yellow fever may be conveyed to a non-immune by the bite of an infected stegomyia fasciata, and that fomites play no part in the transmission. They made a number of observations in regard to the habits of mosquitoes and experimented upon methods for protection from them, and for their extermination. They consider sulphur dioxide the best gaseous insecticide with which they worked. The party undertook some filtration experiments with malarial blood, with the hope that they might elucidate their findings with the blood serum of yellow fever. It was found that the tertian and æstivo-autumnal malarial parasites would not pass through a Berkefeld filter. The serum obtained from a case during the chill of tertian fever, after filtration produced, however, a chill, fever, and sweating when injected into another man. The result was not obtained by serum drawn from a patient during the decline of an æstivo-autumnal paroxysm.

The authors consider that they demonstrated a poison in the blood of a man during the chill of tertian malaria, but that their data are too limited to consider it as the malarial toxin.

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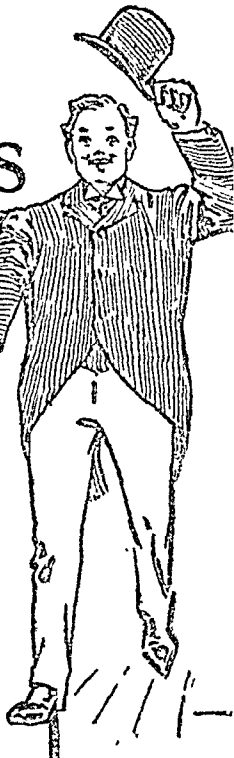
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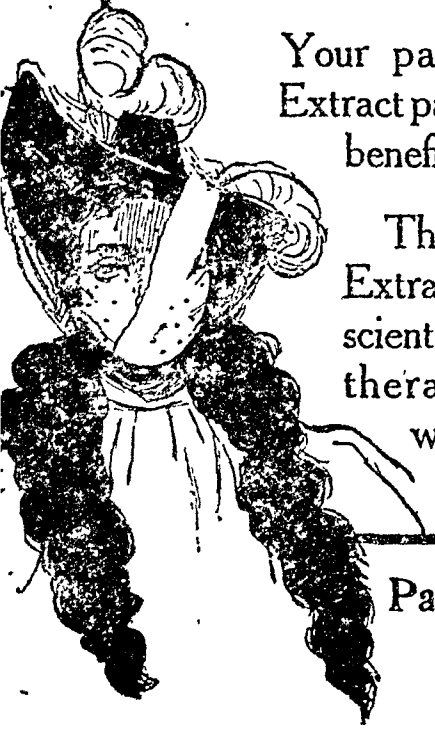
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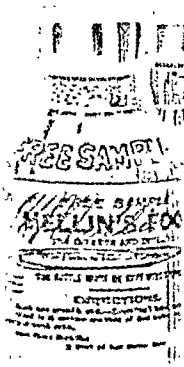
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THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES.

OCTOBER, 1905.

CONCERNING SURGICAL INTERVENTION FOR THE INTRACRANIAL HEMORRHAGES OF THE NEW-BORN.[1]

By Harvey Cushing, M.D.,

Associate Professor of Surgery, Johns Hopkins University.

In a recent paper[2] some generalizations were made in regard to the propriety of operative measures for certain maladies, primarily of neurological interest, that are commonly supposed to be beyond therapeutic aid. The cerebral palsies of children were among the conditions then cursorily reviewed, and the belief was expressed, that, at the time of onset, the indications for immediate surgical intervention are as definite in many of these cases as they are when corresponding symptoms are unmistakably the result of a traumatic intracranial hemorrhage occurring in adult life. This expression of opinion has called forth so many inquiries and led to so many friendly comments—not always of approval, it must be said—that I have been persuaded to amplify the original paragraphs and to record in greater detail my few operative experiences in order to make my position in the matter more clear.

For the convenience of clinical grouping, a variety of terms are used to indicate those individuals who give evidence of serious cerebral defects that have been acquired during infancy. In the main, these terms are descriptive merely of a common residual symptom—the unilateral or bilateral spastic palsy—and this means nothing more than that the so-called motor strip of the cortex, to a greater or lesser extent, on one or both sides, has suffered irreparable injury early in life from an intracranial lesion. Although

[1] Presented at the Philadelphia meeting of the American Neurological Association, June 2, 1905.

[2] The Special Field of Neurological Surgery, Johns Hopkins Bulletin, April, 1905.

it must be kept in mind that the spastic paralysis is by no means a necessary consequence of the lesion—for the cortical injury may be limited to an area of the brain remote from the central gyri, and so lead to no motor disability whatever—the integrity of the cortico-spinal efferent path is, as a matter of fact, and for reasons to be given, very apt to be implicated. A small lesion, which in any other situation might pass unobserved, when placed here will later in life give unmistakable evidences of its presence, as is well shown by a case to be mentioned below (Fig. 1). In the more serious injuries both hemispheres may suffer, and if the lesion is widespread, not only may the spastic diplegia, so well depicted by Little in the middle of the past century, be a consequence, but also varying grades of amentia as well.[1]

Inasmuch as most of the cases are not immediately fatal, being serious only from the distressing late consequences of the injury, there is little known of the morbid anatomy of the disease in its inception. It is, however, generally conceded by the many writers on the subject that a cerebral vascular lesion—hemorrhage in a large majority of cases—is the causal factor of the malady of prime importance. This applies to the cases of prenatal as well as to those of postnatal origin, but more particularly to those incident to parturition—the so-called "birth palsies," with which this paper primarily deals. In these the intracranial hemorrhage is usually of venous origin and follows the rupture of some of the delicate and poorly supported venous radicles of the cerebral cortex. Such an injury may be the direct result of undue traumatism during labor or may occur when too great strain has been put upon the vessels by the profound venous stasis of postpartum asphyxiation; just as in later months they may rupture under the passive congestion brought about by a paroxysm of whooping-cough or a severe convulsion. Though Little's three papers, doubtless more than the writings of any of his predecessors, served to call attention to the malady, he had very few pathological data with which to support

[1] It is, of course, presumable that there have been included in this large group of cases several diseases, which, from an etiological standpoint, are quite distinct, but which, being correspondingly destructive to cortical function, leave traces that in later years make them symptomatically indistinguishable For example, should Strümpell's opinion that there is a cerebral infectious process—polioencephalitis akin to the more definite poliomyelitis—be finally established, a large group of cases, particularly those of the infantile hemiplegic type, could definitely be separated Many have already accepted his views On one occasion I performed a craniotomy on a child four years of age in an acute febrile attack, suffering with headache, vomiting, unilateral convulsions, and presenting a bilateral and rapidly forming stasis papilla The trephination was conducted merely for the relief of tension, but with the hope at the same time that the symptoms might be due to a cortical hemorrhage The exposed hemisphere was found tense, œdematous, and with a multitude of punctate hemorrhages scattered over the various convolutions I presume that it was an instance of Strümpell's form of encephalitis, and on another occasion I should not hesitate to remove a small piece of the cortex for histological examination The pathology of these conditions may more certainly be learned by early operative intervention than in any other way

his contention, based largely upon clinical histories, that calamities of childbirth were responsible for a large proportion of the crippled and spastic children that had come under his care. This is the more apparent when one realizes how much importance has always been given by writers in all languages to the careful pathological reports which accompanied the papers of Dr. Sarah J. McNutt, published twenty-five years after Little's contributions called attention to the subject. Why such reports have been uncommon is not easy to explain, unless it is that autopsies of infants who have died seemingly from asphyxiation have rarely been conducted with this particular point in view. A few years ago, when interested in making some observations on the circulation of the cerebrospinal fluid, I was privileged by the obstetrical department to study, for the purposes of that investigation, quite a number of infants who had been stillborn or who had died during the early hours or days of life, and found, to my surprise, that a considerable percentage of them had died with an intracranial hemorrhage. This, too, has been the experience, I believe, of most pathologists, though records of such findings are astonishingly rare. Whether these cortical hemorrhages in the cases which I examined had been the cause of death could not, of course, always be told, though in some of them the effusion of blood was so great that little doubt was felt but that it must have played an important part in the asphyxiation which the clinical histories recorded. In one of the cases a large, thick clot was found overlying an entire hemisphere, and practically confined to one side, a condition which has been present in two of the cases on which I have since operated. In other instances the extravasations were smaller. One of them was in the cerebellar fossa, associated with a fissure in the occipital bone. In another case the chief distribution of the clot was over one of the occipital lobes.

When one considers what an immense amount of traumatism the infant's head must necessarily be subjected to, particularly should the child be large and the firstborn of an elderly mother, it is little wonder that lesions of this kind occur. And this even without the additional strain that may be occasioned by the instrumental delivery which a narrow pelvis or inelastic soft tissues may ultimately necessitate. The infant's brain itself is doubtless fitted by nature to withstand, without injury or shock, a degree of moulding and manipulation which could not be borne by an adult, fully developed, and medullated brain. And furthermore in cases of traumatic œdema, or of actual hemorrhage, the distensibility of the partially membranous skull will accommodate effusions of such extent that were they to take place in the closed adult skull in proportionate degree, they would rapidly prove fatal. It has been my experience that of the adult cases with an equally extensive hemorrhage only those recover in which there is an associated great fragmentation of the cranial vault, and this, by allowing for some cranial

expansion, releases the brain in a measure from the compression effects that otherwise would be produced. It is probably this distensibility of fontanelle and skull which lets many of these infants escape death, for, being at this time little more than spinal animals, they may live with their bulbospinal axis alone, even in case the cerebrum is thrown in large part out of function. The early days of life, however, when medullation is rapidly taking place, are, so far as cerebral development goes, possibly the most important of all, and it is natural to suppose that the pressure of a clot which may be long in absorbing, or which organizes slowly, will, through a local compression anæmia, prevent the normal development and lead to the cerebral abnormalities that we recognize as the late evidences of the lesion.

It is an impression gained from my own studies, and, I believe, one that has been advanced by others, that the vessels most likely to rupture are those which ascend over the cortex and enter the longitudinal sinus from the midcerebral region. These veins, which lie in the subarachnoid spaces, and for the most part are protected from pressure by following the sulci as they ascend toward the midline, leave this protection when near the vertex, and apparently with no support whatever cross the subdural space to enter the dura at the outer edge of the sinus. In the adult brain there are many adhesions between these cortical vessels and the adjacent dura, particularly where this underlies the lateral expansions of the longitudinal sinus, for here the membrane is punctured in many places by the granulations of Pacchioni. In infants, however, as is also the case in the lower animals, there are no such adhesions, the unsupported vessels being the only connection between the leptomeninges of the hemispheres and the dural sheath underlying the sinus. This can easily be seen by removing the infant brain and calvarium in one piece and by allowing the former to sag away from its bony and membranous covering.

During parturition there is presumably always some overlapping of the parietal bones, owing to the lateral compression of the skull, a condition thought to be more marked with the after-coming head or in the precipitous expulsion of a premature child, whose cranium is less resistant to pressure It is a condition, also, which must necessarily be accentuated by the lateral application of forceps.

Although the cranial moulding may take place to a considerable extent without putting any great strain on these lateral cerebral veins, any undue overlapping could readily tear one or more of them away from the sinus. This, I think, is the more common form of injury[1] and possibly accounts for the fact that the primary

[1] In two of the cases that I have examined I have satisfied myself that such ruptures were present A positive statement, however, cannot be given even for these cases, since the dissection and exposure, difficult enough under any circumstances, owing to the delicacy of the vessels is the more so when they are obscured by extravasated blood One of them was Case IV. of my series Cases are on record in which the longitudinal sinus itself has been lacerated

effusion and so the thicker portion of the clot is commonly median, and, in consequence, implicates primarily and most seriously the centres for the lower extremities. The rupture of the vessels may take place only on one side, and I am of the opinion that the extravasation is usually limited to one side of the falx, though, indeed, a bilateral lesion is common enough, as the many patients with diplegia would indicate. Intracranial bleeding may, of course, result from lesions other than the laceration of veins from overlapping. Fractures, particularly basal ones, may be followed by a serious hemorrhage, and in these instances, as Case III. illustrates, the extravasation may not find its way to any great extent on to the outer surface of the hemispheres, but may infiltrate and affect primarily the basal ganglia. It is possible that injuries of this sort, particularly when the thalamus is involved, may be the ones which lead to the extraordinary condition of double athetosis; for in these cases so far as my experience goes—I have seen but one of them at operation—the hemispheres are absolutely normal in appearance, and it is noteworthy, as well, that in them epilepsy is a rare sequence and intelligence fairly well preserved.

In the numerous and valuable monographs that deal with the cerebral palsies of children—and particularly is it true of the birth cases—there is a remarkable absence of any description of the early symptomatology of the lesion; its consequences alone are given in detail. This may possibly be due to the fact that there is a silent period, so to speak, in the malady, during which the child "improves," and after some months, when it becomes evident that normal cerebral development is not taking place, the patient has very commonly passed out of the obstetrician's hands. So that beyond the account of the abnormal labor there are few data from which we may construct the symptomatology of the early intracranial lesion.

Judging from the patients whom I have seen, however, the condition need not be difficult of diagnosis. Had there been in any of the cases, a serious doubt as to the actual nature of the lesion, it might have been decided by a lumbar puncture, for when there has been any effusion of blood into the subdural spaces, free corpuscles will soon after be found in the fluid of the lumbar meninges. This has proven of great value in determining the presence of subdural bleedings in adult cases, and in infants might serve to differentiate between the hemorrhagic and infectious cases were diagnostic difficulties present. The history of the labor is, of course, all-important; also the degree of postpartum asphyxiation. Among the objective signs should be placed first and foremost the condition of the fontanelle. The membrane is bulging and in some instances may be so tight as to show no pulsation. In two of the patients its bony outlines, in consequence of the tension, could not be palpated,

and it has several times been learned from parents, whose hemiplegic children have been seen for the first time some years after birth, that the fontanelle could not be detected during the first days or weeks of life. Convulsions, of course, are frequent, and particularly if they do not appear until several days after birth they may be more or less unilateral in character. There may be also an undue reflex activity, the child starting or twitching in a convulsive way in response to any sensory stimulus. Ocular palsies or differences in the size of the pupils apparently are not uncommon, and in severe cases there may be symptoms referable to the medulla, as alterations from the normal in the cardiac or respiratory rhythms. In the early days evidences of paralysis are rare. There may be no difference observable in the muscular tone on the two sides of the body, even when the hemorrhage is confined to one hemisphere.

Thus far merely the initial lesion and the symptoms characterizing the onset of the disease have been briefly considered, and, though the critical stage of the malady, it is, to any but the obstetrician, its least familiar period. In its fully developed and terminal aspects the disease is better known—far too well known to neurologists, orthopedists, or attendants upon the asylums for feeble-minded children. A supposed restoration to health may gradually follow the first few precarious days of life, and not until some months have elapsed do the late manifestations of the disease begin to unfold and to bring to light the spastic palsies, or blindness, or deafness, or feeble-mindedness, or, in severe cases, even complete amentia.

There is one very serious sequel which frequently (two-thirds of the cases according to Gowers) accompanies the mildest grades of the disease, even when intelligence may have in nowise suffered. This is epilepsy; and the convulsions, often Jacksonian in character, are akin in all respects to those which follow the cortical changes resulting from cranial injuries with hemorrhage in adult life, and the late evidences of the lesion may have much the same gross appearance.[1]

[1] It may be said, too, in passing, that these cases of epilepsy associated with spastic palsies in children are almost as likely to be bettered by properly conducted craniotomies as are the post-traumatic epilepsies in the adult. During the past few years I have explored about thirty of these cases, and the abnormal conditions encountered defy description. I have seen a right-sided porencephalic cyst with spastic hemiplegia and contractures in a patient whose intelligence was practically normal; a microcephalic child with complete amentia, in whom both hemispheres were exposed, disclosing nothing that could deserve the name of a convolution. In some instances the dura and the cortex may be free, in others densely adherent, and at times there may be large, new-formed, venous sinuses over the lateral surfaces of the dura. In one such case a vertically placed sinus overlay the central area and was lined by Pacchionian granulations. There may be large areas of convolutional atrophy, and the situation of these areas is usually shown before the operation by configurative alterations in the shape of the skull. Twice I have seen in epileptics a flattening of this sort over one occipital lobe: they were cases of birth hemorrhage with homonymous hemianopsia. One of them was operated upon, disclosing a sclerosed and atrophied lobe with adhesions. There may, however, in these patients be no adhesions whatsoever between brain and dura, but merely some thickening of

It is this complication which brings many of these patients to the surgeon and gives him an opportunity of seeing at first hand and during life the extraordinary variety of the cerebral lesions that result from the original hemorrhage. In the adult, however, such a destructive cerebral lesion as may take place in infancy and still be compatible with life could hardly occur, and it is possible that the more firm and resistant character of the fully medullated brain may tend to check a venous extravasation which in the infant might have become huge and perhaps have been the forerunner of porencephaly.

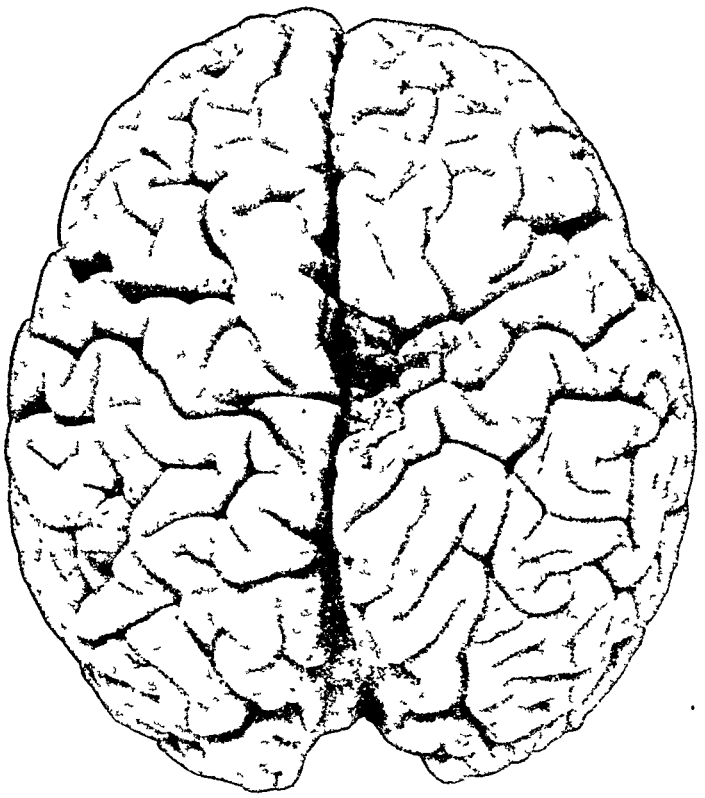
The brain of one case, which I have had an opportunity of studying postmortem, is deserving of mention in some detail, as it shows a lesion, resulting from a birth hemorrhage, which is limited to the motor area of the lower extremity on one side. The patient was a colored girl, thirteen years of age, the firstborn, who had been delivered with instrumental aid. Convulsions had occurred soon after birth, and she had suffered from epilepsy from infancy. The attacks were typically Jacksonian in character and always began in the left leg, which was spastic and much smaller than the other in all of its measurements. The child seemed normally bright and intelligent. She died in the hospital of an intercurrent trouble while awaiting operation. An area of sclerotic atrophy, as shown in the accompanying photograph (Fig. 1), was found, limited to the motor area of the left leg. It is here that lesions would naturally be expected to occur as a result of a rupture of the veins entering the sinus. A similar lesion, if bilateral, would have led to the paraplegia so commonly seen, or to the diplegia of Little if the hemorrhage had been extensive enough to reach to the motor centres of the arms. The remarkable way in which the cortical lesion may be confined to the Rolandic convolutions on the two sides is well demonstrated by the careful study, made in 1885, by Dr. Welch, of Sarah J. McNutt's case.

It is the absolute hopelessness of any form of therapy in these late stages—other than tenotomies to relieve the spastic contractures, or the surgical attempts to check the epileptic seizures—that have led to the despairing words with which the best-known writers on the subject dismiss the therapeutic section of their papers. In his valuable monograph Freud gives his own and a distinguished American neurologist's opinion as follows: "Die Therapie der In-

the pia-arachnoid, with an increase in the amount of fluid filling the sulci. In one patient seen with Dr. Osler and Dr. Thomas, of double athetosis associated with extremely painful cramps, both hemispheres were exposed at different sittings, and the cortex on each side was found to be perfectly normal in appearance, and to give clean-cut responses upon faradization of the precentral convolution. A large portion of this convolution was removed from one side, with a cessation of the cramps, but without affecting in any way the athetoid movements, and giving merely the usual temporary palsy of the extremity. Many other instances might be cited. In the whole series there have been no fatalities. The outer wall of the tense porencephalic cyst in two cases has been removed without appreciable symptoms. Dr. Starr, however, has recorded a sudden fatality following such a procedure.

fantilen Cerebrallahmung ist ein armseliges und trostloses Capitel sowohl an sich als im Vergleiche zu dem mächtigen klinischen Interesse, welches diese Affectionen erregen. Hier gilt, leider unwidersprochen, der Ausspruch von Allen Starr: 'Hemiplegia, sensory defects, and imbecility, occurring with or without epilepsy in children, are chronic diseases incurable by medical treatment.'"

FIG 1



Photograph of the brain of an epileptic, in which the scar resulting from a birth hemorrhage is seen occupying the upper end of the right precentral gyrus (motor area of the left lower extremity) The entire convolution is smaller than the corresponding one on the left Symmetrical incisions, due to removal of tissue for histological study.

It is hoped that in some instances, just as after serious cranial traumatism in the adult, the late results of the meningeal hemorrhage in these cases of so-called birth palsies may be warded off by an immediately conducted operation.

My four operative experiences are represented by the following cases:

CASE I.—The patient, a male child three days old, was seen in consultation with Dr. Dabney on the afternoon of August 22, 1903.

The child was the first of twins, born at full term of a primipara thirty-five years of age. Though a normal head presentation, the labor was slow and prolonged, and low forceps were finally applied. The delivery was easily accomplished without the exertion of force. The child was deeply asphyxiated and it was some time before it could be made to take a breath. Its twin brother, born a few moments later, was normal in every respect. The first child did very badly for the first two days. It could not suckle, and would only occasionally swallow the drops of milk which were put in its mouth. It had a peculiar dusky, cyanosed appearance. There had been several convulsive seizures.

The symptoms were the more obvious, as we had the normal child for a control. The fontanelle was tense and without pulsation. The left eye was kept closed and the pupil on this side was dilated, being twice the diameter of that on the right side. Both reacted to light, the right more than the left. The winking reflex was present on both sides, but only when the lashes were touched. The supraorbital reflex was present, more active on the left. The child could move its arms and legs, and there seemed to be no spasticity nor difference in muscular tone on the two sides of the body. The convulsions had not been unilateral.

Contrasted with its twin, whose pulse was 110 and regular, this child's pulse was considerably slower, having an irregular action of about 92 beats per minute. The respiration, which had been rather irregular and shallow from the beginning, had on this last day assumed something of a Cheyne-Stokes type of rhythm, with periods of apnœa. The child was prepared for immediate operation.

Operation. August 22d, 6 P.M. After the head was shaved and prepared, a few drops of chloroform were administered and a horse-shoe-shaped incision was made through the skin just within and concentric with the border of the left parietal bone. An osteoplastic flap was then raised, including almost the entire parietal bone. The bone was cut through with a stout pair of blunt, curved scissors, very close to its thin and serrated edge. On elevating the bone-flap a tense, dark, plum-colored dura was brought into view. When this membrane was opened and reflected, a clot about 1 cm. in thickness, apparently covering the entire hemisphere, was disclosed. This clot was lifted off in large pieces from the exposed area of the cortex with a blunt instrument, and a large amount of clotted blood which extended over the frontal and occipital regions was irrigated out from under the dura with warm saline solution. The wound was closed without drainage.

Despite its enfeebled condition, for it had taken practically no nourishment during its three days of life, the infant stood the operation remarkably well. A small subcutaneous infusion of salt solution was given, and two hours after the operation the child took some

nourishment from a bottle by sucking the nipple for the first time since its birth. Though there seemed every prospect of a recovery, the infant suddenly died during the night, about eight hours after the operation.

No examination was allowed, and though it would have been interesting to have learned whether there was an extravasation over the opposite hemisphere, it seems quite likely, in the light of the last case in this series, and in view of the unilateral palsy of the third nerve, that an effusion, even were one present on the right side, was less extensive than the one which had been disclosed at the operation.

CASE II.—The patient was first seen March 13, 1904. It was born of a primiparous parent a week previously, on the 6th of March. It had been a tedious breech presentation, with manual delivery. The head had been released with some difficulty. A condition of "pallid asphyxia" was present at birth, but in a few moments, after a hot and cold plunge, the child cried vigorously. There was no overlapping of the cranial bones or other obvious effects of traumatism. During the following night the child kept both of its eyes open most of the time, and for the next five days nothing unusual was noted, except this apparent wakefulness and some stupidity in nursing. The child was not fretful, its cry was strong, and it took nourishment from a bottle fairly well. It had begun to gain slightly in weight. On the fifth day there was a slight elevation in temperature (101.6°), for which there was no obvious cause. The pulse was somewhat slower than usual, registering between 80 and 100 beats per minute.

On the sixth day, Saturday, March 12th, at 9.15 A.M., convulsions first set in, the first spasm lasting perhaps two minutes. During the day there were five other convulsive seizures of varying type and of increasing severity. In the more severe seizures the most decided twitching occurred in the right arm and leg. Some of the convulsions were confined to the face and head, the arms and legs being quiet. In one of the attacks the eyes were crossed, with smacking of the jaws, and with the head thrown to the left, and some rigidity of the neck. The child would sleep after the convulsions, occasionally with its left eye tightly closed and the right partly open. At 11 P.M. there were several severe and repeated convulsions, with some opisthotonos and with a sharp cry at the termination of each.

On Sunday, March 13th, there were eleven convulsive attacks at varying intervals, several of which I observed, when late in the day the child was seen in consultation with Dr. E. F. Cushing and Dr. Crile, of Cleveland. Most of the seizures were bilateral in character, though some of them seemed to us more marked on the left side. There was a constant inequality of the pupils, the left being somewhat dilated, or the right contracted, except during an

attack, when both of them were wide. The fontanelle was not as tense as in the previous case, and, though slightly bulging, its bony outline could be made out.

Monday, March 14th. Up to 9.30 A.M., an hour before the operation, there were seven convulsive seizures, more severe than on previous occasions, and associated with occasional cyanosis. They were for the most part left-sided, and were accompanied with a conjugate deviation of the eyes to the left. One of the spasms lasted five minutes. After the attacks a clear, non-frothy saliva with mucus would pour in considerable quantities from the child's mouth.

The operation, at which I was assisted by Dr. Lower, was performed at 10.30 A.M. Every effort was made to preserve the warmth of the patient and she was wrapped thickly in cotton. The head was shaved, and, as in the case above described, under chloroform anæsthesia, the parietal bone was turned down with its overlying soft parts, this time on the right side. The dura was found to be somewhat tense, and though not deeply plum-colored, as when it covers an extensive clot, it nevertheless showed the presence of an underlying extravasation. The membrane was opened, turned down, and the free blood and small separate clots which were scattered over the surface were irrigated away with warm salt solution. The wound was closed without drainage.

The child slept for two hours after the operation, awoke, cried vigorously, and took its nourishment eagerly. The convalescence was without incident. The wound healed by primary union, the slight pyrexia, which had been present for some days, subsided, and at no time following the operation was there a suggestion of any further convulsive seizures.

On March 28, 1905, the attendant writes me: "The child was a year old March 6th; it stands, takes a few steps, says a word or two, and is a fine specimen of wholesome babyhood."

CASE III.—The patient, with the following history, was seen for the first time October 11, 1904, in consultation with Dr. J. Whitridge Williams. A large baby, a girl, was born October 3d, after a difficult labor lasting forty-eight hours, which was finally terminated by a low forceps operation. At birth the child was profoundly asphyxiated, and only after vigorous efforts was spontaneous respiration established. There was a small abrasion, evidently made by the instruments, over the frontal region. This little wound bled considerably, healed up partially, and later on began to bleed again, so that it was thought the child might possibly be hæmophilic. On the day following birth the right eyelids began to swell somewhat, and there was considerable œdema in the surrounding tissues. On the third day there was an extreme protrusion of the eyeball (Fig. 2), which appeared in the course of a few hours. For several days after birth the child nursed fairly well at

the breast, but later on it refused or was unable to suckle, and nursing on October 8th had to be discontinued. The child became dull and stupid and would not swallow even the nourishment put in its mouth. Its respirations were very irregular. On the morning of October 10th they became markedly Cheyne-Stokes in character, and in the afternoon there was great restlessness and convulsive movements of the arms and legs. Twitching, particularly of the right hand, was noted, and some rigidity of the legs was first observed on this date. The nurse had noted that the child's pulse often registered below 100 beats per minute. The fontanelle was exceedingly tense.

FIG 2



CASE III.—Photograph of the nine-day-old comatose infant, taken the morning of operation. Extreme degree of ocular proptosis, due to cavernous sinus thrombosis

When first seen by the writer it hardly seemed possible that the child could survive, and indeed its life had been for a day or two despaired of. The most startling feature was the extraordinary protrusion of the right eyeball with corneal opacity and hemorrhagic œdema of the lids and conjunctiva. The fontanelle was bulging and so tense that its bony margins could not be made out; there was no pulsation. The radial pulse was very irregular in rhythm and quality. The respiration was irregular, with long periods of apnœa.

There seemed little question as to the presence of an intracranial hemorrhage, though considerable doubt existed as to its exact situation. The history of a sudden non-pulsating protrusion of the

eyeball, with great œdema of the lids and conjunctiva—a condition rarely if ever met with except after cavernous sinus thrombosis—made it seem most probable that a basal fracture had occurred with resultant laceration of this vessel, which had not thrombosed and produced the proptosis until the third day.

It seemed to us both that a craniotomy offered the only hope of recovery. The child was transferred to Dr. Williams' department

FIG 3



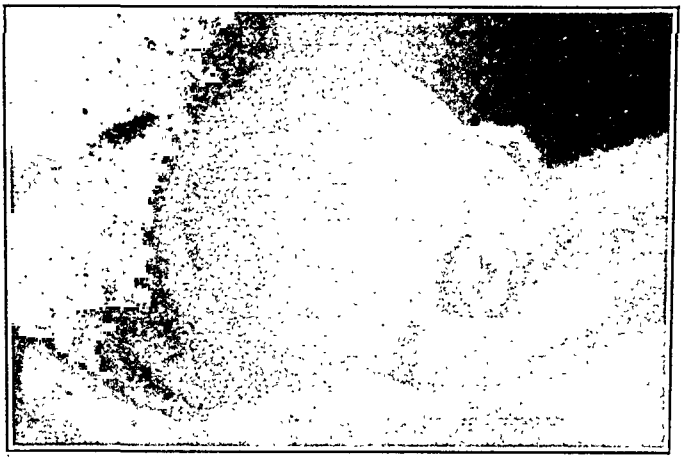
CASE III.—View of cranial vault, to show scars of the bilateral operation.

at the hospital, where an operation was performed early on the following day, October 12, 1904.

As in the previous cases, an osteoplastic flap, including most of the parietal bone, was elevated. This was first done upon the right side. The dura was found to be extraordinarily tense, and, in the endeavor to reflect this membrane by an incision concentric with the osteoplastic flap, some cortical injuries were unavoidably made, owing to the great cerebral tension, which caused the brain imme-

diately to bulge through the small primary opening. This protrusion was so extreme, and the soft, jelly-like infant's brain so easily lacer-

FIG. 4



CASE III.—Lateral view, to show size and position of one of the symmetrically placed osteoplastic flaps.

FIG. 5



CASE III.—Photograph, taken during sleep, of patient two months after operation, to show *complete retrocession of exophthalmos. Contrast with Fig. 2.*

ated, that it was impossible to elevate the temporal lobe sufficiently to discover whether or not a clot was present at the base as a result of the presumed lesion in the cavernous sinus. The operator had to be satisfied with nothing more than washing off from the cortex of the numerous old blood clots which were present on the exposed surface, as well as those which could be liberated from below by such slight dislocations of the hemisphere as was possible without occasioning further cortical lacerations. With the removal of these clots and the escape of cerebrospinal fluid, the bulging, though still considerable, diminished to such an extent that the dura could be brought together again by sutures. Being fearful, however, lest the tension was sufficient to break down these sutures and lead to a hernia cerebri, or, worse, to a fungus, the desperate nature of the case seemed to justify a similar exposure of the other hemisphere. Consequently, a parietal flap was turned down in like fashion on the left side, and a similar procedure carried out, with the washing away from the surface of numerous blood clots and the escape of cerebrospinal fluid. This sufficed to diminish the tension to such an extent that the fontanelle became depressed, and the brain no longer tended to protrude through the dural openings. These were carefully approximated by sutures, the bone-flaps were replaced, and the scalp wounds were closed without drainage.

The child, contrary to our expectations, and almost against our desires, immediately began to improve. By the following day nourishment was well taken from a bottle and she soon began progressively to put on weight. Both wounds healed by primary union (Figs. 3 and 4). It became evident after a few weeks that the cavernous sinus was once more becoming re-channelled. Ultimately, the circulation from the eyeball became re-established, and the exophthalmos gradually disappeared (Fig. 5). It has, however, probably left the eye completely blind.

It is, of course, difficult to tell, before a few years have elapsed, what will be the mental status of the unfortunate child whose life seems to have been saved in this way. At the present time, however, it seems to be developing normally, and is a natural and healthy-appearing baby, with no evidences of motor disability.

CASE IV.—The patient, a second child, was born on the evening of Tuesday, February 14, 1905, after a brief and rather precipitous labor. It was first seen three days later in consultation with Dr. Iglehart, who, though not present at the labor, saw the child soon after birth, and at that time believed it normal in every respect. On the following day, however, his attention was called by the nurse to a swelling which had appeared over the left parietal region. This was somewhat tense and fluctuant, and presented the appearance of a cephalhæmatoma. This swelling during the day, February 15th, and on the following day, increased rapidly in size, until by

the evening of the third day it had become as large as a hen's egg. Beyond this, nothing was thought to be wrong with the child until Friday, February 17th. On this day it was first noticed that the left pupil was larger than the right, and that the pulse had become slow. The nurse also noted that the child had become stupid in taking its nourishment. Fearing that the symptoms indicated an intracranial hemorrhage, Dr. Iglehart made every effort to secure an early consultation; there was an unfortunate but unavoidable delay of some hours, and during the interval the child had become very much worse.

When first seen the large, tense swelling on the left side of the head, its base corresponding exactly in size, position, and shape to the left parietal bone, gave the child a very remarkable appearance. The swelling projected from the surface of the head fully 5 cm. It was pulsating. Pressure upon it did not seem to affect the infant in any way. The left pupil was dilated and the left eyelids kept closed. The eyeballs were somewhat prominent, though there could be said to be no definite exophthalmos. The eye-grounds were not examined. The superficial veins radiating from the angles of the eyes, and particularly those running over the scalp, were so much dilated as to be visible at a distance. There were numerous small, punctate hemorrhages in the skin over the neck and chest. The child's right side was possibly more rigid than the left, but this could not be positively stated. There had been one severe general convulsion about four in the afternoon. The child had taken practically no nourishment for twelve hours.

As rapidly as possible preparations were made for the operation. While the head was being shaved the infant had a second general convulsive seizure, accompanied by several sharp cries. Its general condition was very unfavorable.

Operation, February 17, 1905, at 10 P.M. The head was carefully shaved and prepared, every effort being made meanwhile to sustain the infant's bodily warmth and strength. Chloroform was used as the anæsthetic, a few drops only being required. A needle was first inserted into the cephalhæmatoma and dark fluid blood flowed out of it. An incision was then made about its base, concentric with the left parietal bone. An amount of clotted and dark fluid blood which must have measured two or three ounces was evacuated. This clot lay between the epicranium and the denuded shell of the parietal bone. There was no indication of any connection between it and the intracranial contents. After the evacuation of this clot and exposure of the parietal bone, the latter was turned back almost in its entirety as in the preceding cases. As was expected, a tense, plum-colored dura, exactly similar to that seen in Case I., was brought into view. The dura was opened and a large clot about a centimetre in thickness, which seemed to cover the entire hemisphere, was disclosed. Much of this clot escaped as soon as the opening was

made, and other large pieces of it were lifted off from the surface with a blunt instrument and removed.

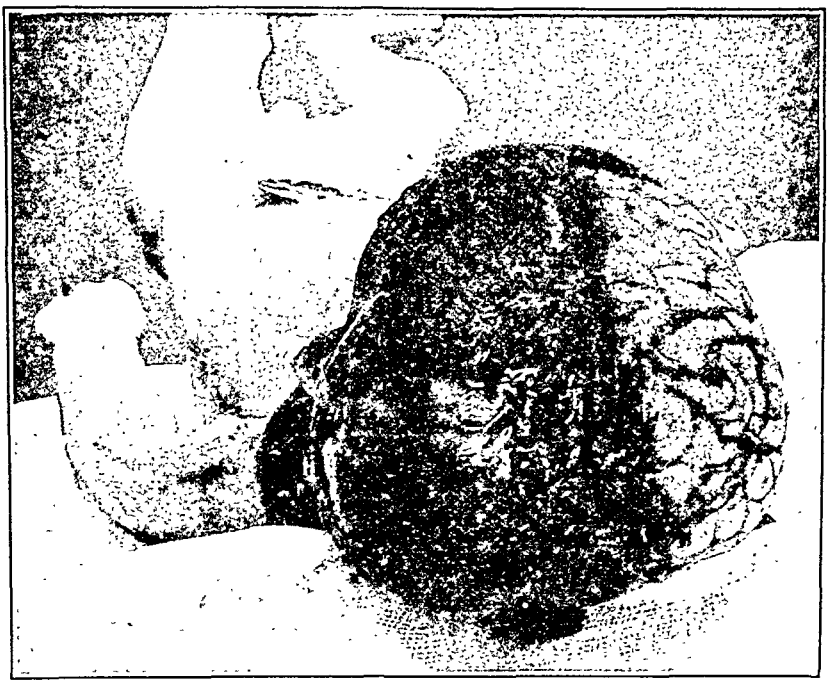
Possibly I should have been satisfied with the relief which this partial operation would have afforded. It doubtless would have been wise to have postponed further interference to another and later occasion, but the child's condition seemed to justify an effort to more thoroughly remove the clotted blood which was within easy access over the anterior and posterior poles of the hemisphere. While irrigating away some of the blood, the child suddenly stopped breathing. Under artificial respiration the heart beat continued for some minutes, but all efforts to restore spontaneous breathing were unavailing.

Postmortem Examination, February 18th. A careful dissection made it quite evident that the cephalhæmatoma had no direct connection whatsoever with the subdural hemorrhage. It seems quite likely that its formation was due to the fact that during the precipitous labor there was a slight injury and slipping of the epicranium from the parietal bone; this did not lead to an extracranial hæmatoma until the intracranial hemorrhage reached a size sufficient to produce great venous engorgement, and this in turn caused bleeding from the torn venules of the epicranium, which under other circumstances would not have had an intravascular tension sufficient to lead to so extensive an extravasation. The outlines of this extracranial hemorrhage were found to be limited by the attachment of the epicranium at the margin of the parietal bone. There was no extracranial extravasation over the frontal or occipital bones. On removing the calvarium it was found that the hemorrhage was limited to the left side of the cranial cavity, and was almost entirely supratentorial. The cerebrospinal fluid over the right hemisphere and in the spinal canal was somewhat bloody, but there were no clots. The accompanying photograph (Fig. 6), taken with the brain still *in situ* but after removal of the calvarium, shows the widespread distribution of the clot. At one place only, the convolutions are exposed; namely, where the clot had been partially removed at the time of the operation. On clearing away the remainder of the clotted blood, which surrounded the hemisphere like a mould, thickest in the central regions, the cortex was found to be macroscopically uninjured. One of the larger cerebral veins was found torn away from the dura at its point of entry into this membrane near the longitudinal sinus.

It seems needless to add anything to the story which these cases tell. There are, however, certain points which may deserve mention before closing this paper. One of them is that a new-born child, with proper regard for hæmostasis and careful avoidance of exposure, will stand a cranial operation well. This is contrary, however, to the statements in our text-books; but when one considers what an amount of traumatism every child must have to endure while making

its entrance into the world, it would seem reasonable to expect that a careful operation, provided there is no loss of blood, would be no more serious. An operation could hardly have been undertaken under less favorable conditions than those presented by the third one of these patients. Yet the child was in better condition a few hours after the double craniotomy than before the operation. Further experience alone will determine the best method of treating the patients with extensive hemorrhages such as the first and last of these cases presented. The operation in each instance should have

FIG. 6



CASE IV —Postmortem appearance of brain after removal of calvarium. Note the extravasation over the entire left hemisphere, the convolutions of which are visible in the parietal region alone, where a portion of the clot was removed at the operation. Right hemisphere totally free from extravasation.

been done at least a day earlier, and, as the results show, I should have refrained from attempting too much at one sitting.

An argument, advanced against the possibility of carrying out these operations on the new-born, is that the blood of these infants has such a slow coagulation time that a fatal hemorrhage from the incision would result. This is no more true for cranial operations in the new-born than for those in other parts of the body, and my experience leads me to think that coagulation is as rapid as in the adult, though I have made but few accurately timed tests. It is

worthy of remark in this connection that the effused blood in all of these cases has been found clotted.

It will have been noted that in two of the patients symptoms definitely pointing to intracranial trouble did not appear or were not recognized until several days after birth. A more careful study of symptomatology—and this will surely follow upon the knowledge that something immediate can be done to relieve these cases and possibly to ward off the consequences of the hemorrhage—will perhaps enable us to recognize these conditions earlier.

In three of the cases—those with primary cortical hemorrhages—the effusion was confined to one side. The birth palsies, however, are more commonly supposed to be bilateral, and many of the writers on the subject regard the spastic diplegias or paraplegias as the typical cases of Little's disease, whereas a spastic hemiplegia is supposed to be more commonly of infectious origin. Extensive as it was, however, the effusion in Cases I. and IV. was unilateral, and had these patients survived, unoperated, a spastic hemiplegia probably would have resulted. In Case II., on the other hand, symptoms, appearing on the fifth day after birth with fever and unilateral convulsions, might easily have been ascribed to some infectious process; yet a hemorrhage was found and they immediately abated after the operation.

If it can be demonstrated that a craniotomy on the new-born child, when conducted with due precaution and delicacy of manipulation, is comparatively free from danger—and Cases II. and III. show how well such operations may be tolerated—I believe the immediate risk of death and the sorry late consequences of meningeal birth hemorrhages may be avoided in many cases by surgical interference. It is reasonable also to suppose that these explorations will at the same time lead us to a better understanding of the varying pathological features of this group of diseases in their early stages.

INTESTINAL PERFORATION IN TYPHOID FEVER IN EARLY LIFE.[1]

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The occurrence of typhoidal perforation in children deserves consideration, first, on account of its supposed rarity, and, second, on account of the difficulty of recognition which is often presented and which seems to me to be largely peculiar to this time of life.

As to the *rarity* of its occurrence there appears to be little variation of opinion among many of the leading text-books. That the accident

[1] Read before the Association of American Physicians, May, 1905.

is uncommon, although just to how great an extent, is a matter regarding which statistics vary. Marfan[1] says that perforation in children is almost never observed. Baginski[2] speaks of it as uncommon, and has seen but 2 cases. Rotch[3] considers it exceedingly rare, and Holt[4] has found but 12 instances in 1028 reported cases of typhoid fever in children. As 4 are said to have recovered we are uncertain of the diagnosis in these. Heubner[5] has never seen a case. Wilson[6] considers it exceedingly rare, and Koplik[7] and Gerhardt-Seiffert[8] are of the same opinion. Henoch[9] has seen it but once in all his cases. Curschmann[10] speaks of perforation as rare in children.

On the other hand Ashby and Wright[11] have met 4 cases, and Méry,[12] although admitting its great rarity, thinks that this has been somewhat exaggerated. He says that at least 14 or 15 cases have been reported from the Children's Hospitals of Paris during the last six years, quotes 26 perforations in 1506 cases collected by Setbon, and says that Murchison, in 73 reported perforations, found 10 in children under ten years of age and 8 in those from ten to fourteen years.

Journal literature, too, although in general speaking of the rarity of perforation in early life gives somewhat varying statistics. Roemheld[13] found 3 perforations in 117 cases, and Montmollin[14] reports 7 cases, 3 of which, however, recovered. Morse[15] reports no perforations in 234 cases, and only 4 in 735 cases published by eight observers whom he quotes. Fitz[16] found 7 perforations among 192 children, and Adams[17] 11 perforations in 337 cases. Rennert[18] gives 4 perforations out of 471 cases, and Schulz[19] 8 cases of perforation in children.

One of the most valuable contributions to the subject is that of Elsberg.[20] Out of 289 collected cases of operation for perforation in typhoid fever, 25 occurred in children less than fifteen years of age. He gives a useful analysis of the symptoms exhibited in these and concludes that they do not differ materially from those occurring in adult life. This article deals so thoroughly with the

1 Traité des maladies de l'enfance (Grancher, etc), 1897, vol i. p 321).
2 Lehrb der Kinderkr , 7 Auf , pp 201, 211. 3 Pediatrics, 1901, p. 451.
4 Diseases of Infancy and Childhood, second edition, p 1054
5 Kinderheilk . vol i. p 508
6 Cyclopedia of the Diseases of Children. Keating, vol. i p. 408
7 Diseases of Infancy and Childhood, p. 173
8 Lehrbuch der Kinderkr., p 175. 9 Kinderkrankheiten, 8 Auf., p 780
10 Nothnagel's Encyclopedia, p 333.
11 Diseases of Children, second American edition, p 283
12 Traité des maladies de l'enfance (Grancher, etc.), second edition, vol. i p. 495
13 Jahrb f Kinderheilk , vol. xlviii. p 207.
14 Thèse, Neuchatel, 1885. 15 Boston Medical and Surgical Journal, Feb. 27, 1896
16 Transactions of the Association of American Physicians, 1891.
17 Archives of Pediatrics Feb , 1904 18 Deutsch. med. Wochen , 1889, p. 1063.
19 Jahrb der Hamburger Stadtskrankenanstalten, 1889, vol. i.
20 Annals of Surgery, July, 1903

literature of the subject that it is not necessary to enter more fully upon it in this connection.

That typhoid perforation should occur less frequently than in adults is in accord with the characteristics of the lesions of the disease in early life. In the fœtus typhoid ulceration is uniformly absent. In infants it is exceptional that it is at all extensive or severe. As age advances it grows more common, but is generally less frequent and less severe than in adult life. Nevertheless, very extensive ulceration can exceptionally occur even in early childhood, as I have seen proven upon the post-mortem table. Consequently, the occasional development of perforation is to be expected as a matter of course, and the older the child the more liable is this to happen.

As my experience has been unfortunate in having met with 6 instances of perforation in children, it may be of service to put these on record. The history of the first has been previously published, but is reported here for the sake of completeness of the group. The others I have not recorded heretofore, although the fact of their existence I had referred to in the discussion of a paper by Dr. J. H. Jopson.[1] The surgical aspects of the sixth case have been alluded to briefly by Martin and Pennington.[2] For various reasons none of the cases had a leukocyte count made when perforation was suspected. It is doubtful, however, whether this, if done, would have aided much in the diagnosis.

Case I.—Lœila B., aged ten years, entered the Children's Hospital of Philadelphia, February 26, 1897, on about the sixth day of typhoid fever. During her first two weeks in the hospital she passed through an ordinary attack of the disease without abdominal pain or tenderness and without diarrhœa until March 12th, when two watery movements occurred. On the evening of March 15th, approximately the twenty-third day of the disease, rather severe pain developed in the right iliac region, yet without signs of any serious trouble being present. The temperature had been approximately 104° unless reduced by tubbing. During the night slight intestinal hemorrhage occurred, with a fall of temperature to 103°. On the following day, March 16th, there was abdominal pain, not very severe, with slight distention of the abdomen, accompanied by tenderness and increased resistance. The pulse had lost in force, and the face had a somewhat anxious expression; yet there was no evidence whatever of collapse, the temperature remaining about the same. Later in the day vomiting began and continued troublesome. The abdomen was more distended and the hepatic dulness was nearly obliterated. The temperature had fallen in the evening to 101°. During the following night the abdomen became more distended and the child died in the early morning of March 17th, the temperature having fallen slowly to

[1] Archives of Pediatrics, March, 1901.

[2] American Medicine, Nov. 21, 1903.

99.6°. None of the ordinary symptoms of collapse had been exhibited at any time. The occurrence of a perforation had been suspected at the beginning, and later believed to be undoubted, but operation could not be performed—for reasons upon which it is not necessary to enter.

The autopsy showed a perforation of the ileum, about fifteen inches above the ileocæcal valve, with extensive and deep ulceration of Peyer's patches.

CASE II.—Charles K., aged eleven years, entered the Children's Hospital on February 28, 1899. He had been ill for four days with fever, malaise, chilly sensations, loose cough, and some pain in the abdomen and hips, "a kind of attack that he had frequently had before." An examination on March 3d showed a slight diminution of expansion of the left side of the chest, with dulness on percussion from the sixth rib downward in front and in the axilla; somewhat feeble respiration and some friction sounds on deep inspiration. Posteriorly there was dulness on percussion over the lower third of the left lung. The spleen could not be felt. The diagnosis of pleurisy or of pleuropneumonia was made.

The boy continued to do well, the temperature remaining elevated from 100° to 103°, and the bowels, at first normal, becoming diarrhœal in character. On the afternoon of March 6th, his seventh day in the hospital, and apparently his eleventh day of illness, he complained of pain in the abdomen and vomited. The symptoms were apparently the result of his having eaten some ice-cream. Vomiting continued all night, and general weakness developed, with feeble pulse, sufficient to make hypodermic stimulation necessary. The temperature arose in the mean time to 105.8° without any previous fall of it. March 7th found him with constant vomiting, a cold and clammy skin, and general abdominal distention, tenderness, and pain. The friction sounds over the lung were less numerous. The condition continued all day, the cause of it being not at all understood. His temperature ranged from 102° to 105°. On March 8th he was better, but the features were quite sunken. The abdomen was less distended, but very tender, and the hepatic dulness not at all diminished. The dulness over the base of the left lung had disappeared. A few small rose spots were discovered on the trunk, suspiciously like the typhoid roseola. There appeared to be some increase of resistance in the right iliac region. The temperature was 106° in the morning of this day and 100.6° in the evening. The existence of typhoid fever was now suspected for the first time, and it seemed very certain that perforation had occurred. No operation could be performed.

At 3 A.M. on the morning of March 9th the temperature dropped to 97.8°, but quickly rose, and at 7 o'clock A.M. it was 101.2°. Failure of strength continued; on March 10th there were sunken features, jactitation, a tense and moderately distended abdomen,

and a greater resistance to palpation of the right iliac fossa. The temperature in the morning was 105.4°, but fell to 102.4° in the evening, when death occurred.

Autopsy showed a general purulent peritonitis; a perforation in the ileum five inches above the ileocæcal valve; other ulcers in the ileum, some apparently in process of healing; evidences of quite recent pleurisy over the right lung, and some soft adhesions over the base of the left lung.

It was evident from the autopsy that the disease was farther advanced than the history indicated, and that the child had been a case of walking typhoid, complicated later by pleurisy.

CASE III.—Celia S., aged twelve years, first seen in consultation with Dr. S. N. Dubin, May 1, 1899. The child was at the time apparently in the fourth week of typhoid fever. Her mind had been clear and her condition good throughout, except for constant tendency to hyperpyrexia still existing, the temperature being often 106° or over. The tongue was moist and there were slight tremulousness and moderate diarrhœa. By May 4th her condition was much the same, except that the diarrhœa had nearly ceased. There was, however, retching when food was given. Later, vomiting began again, but was only occasional, and the child's condition continued no worse. There were at times specks of blood in the stools. At five o'clock on the morning of May 9th she suffered from sudden severe abdominal pains, and a fall of temperature from nearly 107° to 97°. Dr. Dubin felt certain that she had a perforation. I was asked to see her again, and did so in the afternoon, but failed to meet Dr. Dubin, who was unavoidably detained. She had at this time moderate abdominal tenderness on pressure, but no distention or vomiting, and no evidence whatever of collapse or any noteworthy abdominal pain. Her temperature was still elevated. I had heard nothing of her suspicious symptoms, and careful questioning of the parents, who were rather ignorant people, failed to elicit any history of their previous presence. The indications of perforation returned later, and she was removed, on May 10th, to the Methodist Hospital, where I saw her again in consultation with Dr. Dubin and Dr. E. W. Holmes, the attending surgeon. She was now clearly very ill, and her mind was partly clouded. Operation was done at 10 P.M., about forty-one hours after the first symptoms appeared. A perforation was found in the ileum, with several ulcers threatening rupture. Death occurred before morning.

CASE IV.—Frank M., aged eight years, was admitted to the Children's Hospital of Philadelphia January 2, 1903, apparently at the end of the first week of typhoid fever, having suffered from loss of appetite, malaise, slight cough, headache, and slight abdominal pain. Examination showed the child emaciated to some extent, the heart normal, a few rales in the lungs, the spleen

indistinctly felt, rose spots present on the abdomen, and slight gaseous distention. His condition continued fairly good, there being very slight diarrhœa, and the temperature running from 102° to 104° or 105° and making hydrotherapy necessary. On January 14th, the thirteenth day of his stay in the hospital, he was still doing well, although having a rather severe attack of the disease, but at 11.30 P.M. vomiting occurred, the temperature being 104°, and continuing from this to 105° during the night. On the morning of the 15th his pulse was weak and irregular and his abdomen moderately distended and only slightly painful on pressure, but extremely tense. The hepatic dulness was normal. There seemed to have been no sudden development of this condition, and his temperature did not fall below 101.2° on this date. He vomited again at 9.45 A.M. and at 11.30 A.M. The possibility of a perforation having occurred was considered, but the absence of any evidence of collapse and the slight degree of vomiting and abdominal tenderness and distention, combined with the presence of normal hepatic dulness, made it seem doubtful. There was, however, a suspiciously anxious expression of face. Request was left that Dr. R. Le Conte, the surgeon on duty, examine him when he made his visit, and a message was sent to his parents asking permission to operate if necessary. The child's symptoms grew worse so rapidly that a few hours made the diagnosis practically certain. The distention of the abdomen increased greatly, as did rigidity and tenderness, the liver dulness disappeared, abdominal pain became extreme and continuous, vomiting returned and occurred repeatedly, and the features grew very much sunken. The temperature continued at about 103°, but diminished to 100.4° in the afternoon. Operative interference was absolutely refused by the parents, who removed the child on the following day, January 16th, against advice. He died on January 17th.

Although no autopsy was held, there can be no reasonable question of the diagnosis.

CASE V.—Mary B., aged four years, was admitted to the Children's Hospital of Philadelphia March 8, 1903. Ten days previously she had commenced to suffer from loss of appetite, malaise, headache, diarrhœa, and epigastric tenderness or pain. After a short time the parents noticed that she had fever, and she was then put to bed. During the last three days the temperature had reached 104° and the other symptoms had continued. On admission she exhibited some distention of the abdomen, enlargement of the spleen, roseola, and a coated tongue. The mind was clear; the Widal reaction was positive. She was considered to be probably in the middle of the second week of the disease. The attack ran rather a mild course, the abdomen showing considerable tympanitic distention, the child looking bright, and the temperature gradually falling. By March 17th, the last occasion on which I

saw her, the patient was convalescent, the temperature having been nearly normal most of the time for a week. I give a synopsis of the ward notes made after this date: At 7 A.M. on the morning of March 18th the child vomited food and medicine and had slight abdominal pain, but her temperature remained at 99° and she did not look ill. At 9.15 A.M. she appeared feverish and vomited again. At noon her temperature was 100°. There was no abdominal pain, distention, or tenderness, and the child was sitting up in bed in apparently good condition, At 3 P.M. the temperature was about 103° and there were still no abdominal symptoms.

Shortly after this she vomited again twice, at 3.15 and at 4 P.M. At 6 P.M. the temperature was 105.6°. The mind was clear, but the child looked ill and was somewhat restless. There was no abdominal distention or pain. By 9 P.M. the temperature had risen to 106.6°, the child was growing rapidly worse, and died at six o'clock on the morning of March 19th. Distention, pain, and tenderness of the abdomen had continued to be absent, and there had been no return of vomiting since four o'clock on the preceding afternoon.

The autopsy showed a purulent peritonitis. The ileum exhibited numerous small ulcers near the ileocæcal valve, and in one of these was a minute perforation. The serous layer of the intestine at this point was adherent to another knuckle of the gut, thus partially closing the perforation.

CASE VI.—Esther S., aged six years, seen in consultation with Dr. Joseph Neff April 4, 1903. The child was presumably in the second week of typhoid fever. Rose spots were present. The general condition had been good. About four o'clock in the afternoon of this day she had suffered from quite severe abdominal pain followed by vomiting, and was said to have had decided depression of strength, although without actual collapse. It is not known whether there was any fall of temperature. I saw her at 10 P.M. and found her in excellent general condition, with the pulse of good quality, no anxious expression of face, slight diarrhœa, no abdominal pain, tenderness only on firm pressure, and distention only slight. Vomiting had ceased. The diagnosis of a perforation was considered, but dismissed as too uncertain to warrant operation.

I therefore advised cautious delay, but immediate operation should any of the symptoms return. A telephone message at 9 A.M. on the next day, April 5th, told me that the child had had a good night, that there had been no return of pain or vomiting, and that the abdomen was not distended or tender. Later in the morning, however, at about eleven o'clock, abdominal pain returned, distention became decided, and the child was removed to the surgical ward of the hospital of the University of Pennsylvania. Here the symptoms continued, perforation was suspected, and operation was done by Dr. Edward Martin at about 5 P.M. in

the afternoon of the same day. One perforation was found and two ulcers threatening rupture. The child was in bad condition immediately after the operation, but by the next day all symptoms were favorable. She did well for a time, but evidences of the formation of a pus cavity developed, and, although this was found and evacuated, her strength failed and death occurred seven days after the first operation.

I have referred in opening the subject to the difficulty in the recognition of perforation frequently exhibited in early life. Some of the cases here reported possessed the classical symptoms as usually seen in adults; others certainly illustrated the difficulty to which reference has been made. Case I., although exhibiting abdominal pain, tenderness, and distention, had at first no more of these than is common in cases of typhoid fever with tympanites. There was no initial fall of temperature or evidence of collapse. The symptoms as exhibited could be called suspicious only. Later in the same day I should certainly have advised operation had it been possible to have had it performed, as there was reason to believe that perforation was probably present.

Case II. was absolutely misleading throughout. Even the presence of typhoid fever was not suspected. The child was supposed to be suffering from pleurisy only. The occurrence of vomiting and abdominal pain seemed clearly connected with an indiscretion in diet. It was only on the day following that on which perforation probably occurred that the suspicion arose that something more was ailing him, and not until the succeeding day that the diagnosis of typhoid fever and perforative peritonitis was entertained. The symptoms by this time were sufficiently positive to make operation urgent, although even now the abdominal distention was not great, the only fall of temperature below normal having occurred after the perforation had existed, probably, over two days.

A most unfortunate circumstance prevented a better understanding of the symptoms in Case III. Those at the onset were clearly those of perforations, or possibly of concealed hemorrhage. The failure of blood to appear later in any large quantity in the stools excluded the latter. Within a few hours all symptoms had disappeared to such an extent that, although entertaining the thought of the presence of perforation, based upon the existence of some abdominal tenderness, I did not feel warranted in believing that this had occurred. Had I known of the earlier condition I should, of course, have advised immediate operation.

Case IV. presented doubt only during the first hours of the condition. There was at that time no history of collapse and no more abdominal distention, pain, and tenderness than is very frequently present in cases of typhoid fever. There was also no loss of hepatic dulness. The earliest really suspicious symptom was alteration

of the facies. In a few hours the symptoms were beyond question.

It is difficult to say in Case V. on what a diagnosis of perforation could possibly have been based. The vomiting was only occasional; the complaint of pain had been temporary and not great. There was no collapse, and throughout the twenty-three hours which followed until death occurred there was neither abdominal tenderness, distention, nor pain to guide us. The count of the leukocytes might have been of significance if there had been any suspicion of what ailed the child, but even this is by no means certain.

Case VI. presented elements of great uncertainty. In view of the fact that vomiting and abdominal tenderness and pain are of such frequent occurrence in typhoid fever, I could not feel warranted in making a diagnosis of perforation or in advising exploratory laparotomy, especially when the symptoms had been unattended by any certain evidence of collapse and had so largely disappeared at the time of my visit. In any similar case I should, I think, again follow exactly the course taken in this—viz., delay cautiously until a diagnosis of perforation was more probable. From the point of view of the immediate results of the operation the child did well. Death occurred rather from a sequel.

A CASE OF ACUTE LEUKÆMIA, WITH SOME STRIKING CLINICAL FEATURES. OBSERVATIONS ON METABOLISM IN THIS CASE, AND IN A CASE OF SEVERE PURPURA HEMORRHAGICA.[1]

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My chief purpose in this report is to discuss certain disturbances of metabolism in leukæmia and purpura hemorrhagica that are of much clinical and pathological importance, and to inquire somewhat into the nature of these disturbances, particularly their relation to the hemorrhage that occurs in both these diseases. I shall, however, first mention briefly some striking clinical features in the case of acute leukæmia, which are of interest from an etiological, and particularly from a diagnostic, standpoint.

There was an apparent connection of the beginning of the symp-

[1] Read at the meeting of the Association of American Physicians, Washington, D. C., May 16 and 17, 1905.

toms with the extraction of a tooth, and therefore with the opportunity for infection, a point that is of some interest in relation to the etiology of the condition; and the clinical resemblance of the case to diphtheria and angina Ludovici has serious diagnostic importance. In this instance the possibility that the disease had originated from mouth infection set up through the extraction of a tooth must, however, be dismissed as extremely improbable. The very early appearance of severe stomatitis in many cases of acute leukæmia has led to the repeated suggestion that the disease itself, or at least the rapidity of its progress, is caused by infection, particularly through the diseased mouth; and in a number of instances, as in my case, the extraction of a tooth was apparently directly followed by rapidly progressing acute leukæmia. In the instance that I am describing, however, the actual conditions were almost certainly the reverse of this; that is, the disease itself, in its incipient stage, evidently produced the symptoms that led to the extraction of the tooth.

As to the appearances suggesting diphtheria and angina Ludovici, there is, I think, in systematic writings dealing with acute leukæmia, or in more fragmentary papers, little, if any, discussion of the possibility of mistaking this disease for diphtheria, and there is no sufficient consideration of the possibility of mistaking the disease for simple angina Ludovici without other general disorder. Late in the course of this case it would have been wholly impossible without bacteriological investigation to have stated that it was not diphtheria; and, on the other hand, angina Ludovici seemed, when I first saw the man, to be probably the main condition present until the blood examination, that was made immediately afterward, showed at once that he had acute leukæmia. In the classes in pathological anatomy in the General Hospital at Vienna, specimens from several cases of acute leukæmia have, I am told, been exhibited in recent years with the statement that they had been mistaken for some time for angina Ludovici. This mistake is a natural one, and could scarcely be avoided in some instances until the blood condition became distinctive. Indeed, there is every opportunity for patients with acute leukæmia to develop actual Ludwig's angina, the condition of the mouth that is so common in this disease giving ready opportunity for infection of the tissues of the neck.

Without giving extensive details of the clinical side of the case of acute leukæmia, I would refer to the following points: The patient, a man aged thirty-six years, was seen with Dr. Cryer on January 15, 1901. He gave a good family history, and a personal history that was of no interest until about three weeks before I saw him. For some months previous to this time he had been under severe business and emotional strain, but no results of any consequence had been noted. Late in December, 1900, the first symptoms of importance appeared. He then had swelling of the gum

about the upper posterior molar tooth on the right side, together with much pain, and his dentist extracted the tooth. The fact of interest at this point is that the trouble with the tooth was almost certainly due to leukæmic infiltration of the tissues about it. The history first obtained from the patient apparently indicated the contrary—that the disease in the mouth and elsewhere had followed the extraction of the tooth, for subsequent to its extraction he had local ulceration of the gum, of unhealthy appearance and with necrotic pseudomembrane over its surface, and this ulceration advanced until it involved a large part of the surface of the gums of the left side of the mouth. At this period, too, the man noticed the first disturbance in his general health. The tooth, however, was healthy, and there was at that time no disease of the surrounding tissues, the necrosis having followed the extraction merely because of the disease that had already begun. It seems probable that had this point been ascertainable, it could have been shown in at least a considerable portion of the other similar cases in the literature that the initial symptoms of leukæmic infiltration actually preceded the development of the local opportunities for infection.

Because of the dental condition the patient was seen in consultation by Dr. Cryer a week after the tooth was extracted, and ten days later, after a telephonic communication, Dr. Cryer had him admitted to the private ward of the University Hospital for examination, thinking that the condition of the mouth might be due to an impacted tooth. It was, however, at once apparent that he had serious general disease, and at Dr. Cryer's request I took charge of him. In the period intervening between this time and his previous visit to Dr. Cryer he stated that there had been marked and rapid loss of strength, with a good deal of emaciation and mental depression; and he had also had decided disturbance of vision and hearing, having had difficulty in reading even the large print of a newspaper, and at times in recognizing the members of his family, and having become so deaf as to be uncertain about ordinary conversation. For some days before entering the hospital he had had much pain on attempting to masticate or swallow, and had had a slight, thin, bloody discharge from the nose; he had also become extremely hoarse, and had had difficulty in inspiration. There had been no other noteworthy symptoms. At the first glance the immediate suggestion was that he had a severe diphtherial infection. He was evidently much prostrated; the pulse was rapid and weak; he had marked inspiratory stridor, and his voice was a hoarse whisper; there was a thin, blood-tinged discharge from the nose; a little pseudomembrane was at once visible in the nose, and he evidently had distress upon swallowing. His temperature was 101.2°. General examination at once made the possibility of diphtheria very questionable; it showed the following conditions: He was weak and seemed emaciated; there were small ulcers, with

hemorrhagic bases on the lips and about the nostrils; there was a narrow line of ulceration of the mucous membrane of the upper jaw, most marked posteriorly, but extending to the incisors; there were similar patches of ulceration on the lower jaw, though less extensive, and there were small ulcers beneath the tongue and on the dorsum of the tongue. All these ulcers had hemorrhagic bases, but were not bleeding. Their surfaces were covered with a dirty looking, yellowish-gray pseudomembrane-like necrotic deposit. The gums were swollen and congested, but did not bleed upon manipulation; the tongue was somewhat swollen. The pillars of the pharynx and the walls of the pharynx itself were very much swollen, and the tonsils were so large that there was a space of only half an inch between them. The lumen of the pharynx also seemed very small, as a result of the swelling of the tissues. There was, however, no redness, pseudomembrane formation, or ulceration back of the tongue. The nose was nearly blocked on both sides, the mucous membrane swollen and somewhat congested, and there were small ulcerated areas which showed a pseudomembranous deposit.

The man's neck appeared to be much swollen, particularly on the left side, and the tissues, on the left side especially, felt dense and infiltrated half-way down to the clavicle. Part of this was evidently due to glandular enlargement, one gland, about one and one-half inches in length, being readily felt, as well as a number that were smaller; but the swelling of the neck was so conspicuous as to be evident at the first glance, and it appeared to be due to a considerable extent to diffuse infiltration of the tissues, and not simply to glandular enlargement.

The glands elsewhere could be everywhere felt, and in the axilla several were about an inch in diameter; the spleen could not be palpated, but the dulness was enlarged. Examination elsewhere showed nothing of diagnostic importance. The lungs were decidedly congested at the bases; the heart was a little dilated, the sounds weak, the action rapid. The man had difficulty in reading even the head lines of a newspaper, and he could hear a watch at but six inches distance with the left ear; three inches with the right.

A culture was taken at once, and proved negative for diphtheria bacilli. The resident physician's blood count, which was immediately confirmed by Dr. C. Y. White, showed 466,640 leukocytes; 1,820,000 red cells. The hæmoglobin estimations at this time and subsequently were unreliable because of the immediate clouding that took place.

The examination of stained specimens showed a very definite but somewhat peculiar picture of acute lymphatic leukæmia. I shall not discuss the morphology of the blood, as I have asked Dr. White to include the study of this blood in discussing a series of cases that he is collecting.

The conditions that I have mentioned show that there was a

strong suggestion of diphtheria and also of angina Ludovici in the case. Twenty-four hours after I first saw him diphtheria could certainly not have been excluded without negative cultures; even the definite determination that acute leukæmia was present would scarcely have finally excluded diphtheria, for at that time he had pseudomembrane on both tonsils, and he had the nasal and laryngeal symptoms that are so common in diphtheria.

The further progress of the case was one of increasing prostration, with death two days and a half after I first saw him, from cardiac failure, œdema of the lungs, and sudden collapse. The course was interesting in only two particulars: In the first place, the obstruction of his larynx grew more and more severe, and finally became so marked that the possible necessity for tracheotomy was kept anxiously in view, though this measure would have been most reluctantly undertaken because of the hemorrhagic tendency. So far as I know, marked laryngeal obstruction has not been described in acute leukæmia, though it has repeatedly been seen in chronic lymphatic leukæmia. The laryngeal symptoms were, as was expected, found to be due to extensive infiltration of the larynx, with marked swelling and slight ulceration of both the false and the true vocal cords.

The other point of interest was the hemorrhage. The man exhibited no hemorrhagic tendency, excepting as a result of the second puncture made for study of his blood. This minute wound was followed by oozing of blood in almost a continuous stream for the greater part of an hour; all measures used to stop it were ineffectual, and the amount of blood lost was certainly as much as a pint. This feature is interesting chiefly in relation to the manner in which the hemorrhage was produced. Grave hemorrhage from the same cause has previously been described in rare instances.

A few hours after his admission, when the diagnosis of acute leukæmia had been made, a metabolism experiment was begun. This, unfortunately, could be continued for but one period of twenty-four hours, because it became impossible after this to save his urine, owing to his profound prostration and mental obscurity. In this time, however, sufficiently characteristic conditions were observed.

He took during this period only milk and broth. The nitrogen content and the amount of P_2O_5 in these were determined. I also determined the nitrogen and P_2O_5 in the urine: estimations of the uric acid and purin bases were begun, but, unfortunately, were lost through the mistake of a laboratory servant.

The man's food contained 7.25 gm. of nitrogen, 1.84 gm. of P_2O_5. The urine during the same period contained 29.534 gm. of nitrogen and 3.056 gm. of P_2O_5. While I lost the estimations of uric acid and purin bases, I think I am safe in saying, from the large precipitate produced by ammoniacal silver nitrate, that the uric acid was greatly increased; it was probably above 2 gm., at any rate.

The feces could not be accurately collected, and hence the nitrogen and phosphates in the feces were not determined; there was probably more than a gram of nitrogen in the feces, however. In addition to this, the man lost during this period, as I have stated, a large amount of blood, and this must have contained 4 gm. or more of nitrogen. The balance between the intake of nitrogen and the outgo in the urine alone shows a loss of 22.28 gm., and with the loss from other sources he must in all have had a negative nitrogen balance during this twenty-four hours of as much as 27 gm. The loss of phosphorus, while decided, was not so remarkable as the loss of nitrogen. It was probably over 2 gm.

Such tremendous tissue destruction is a condition that is comparatively rare in other diseases, but it seems to be very common in acute leukæmia, as far as the very few chemical investigations of this disease that are on record indicate. It appears, therefore, to be a very important element in the pathology of the condition. Magnus-Levy, in particular, directed attention to this very remarkable disturbance of metabolism, reporting his own results in three cases, in one of which he determined the nitrogen of the urine, and in the other two the actual balance between the intake and the outgo. In the first of his cases there was a loss in forty hours by the urine alone of 24.8 gm. more than the patient had taken in his food. In addition to that, he lost 3 gm. in the feces and about 17 gm. by hemorrhage, so that in all this patient showed in a period of forty hours a negative balance of nearly 45 gm. of nitrogen. In the second case 85.4 gm. of nitrogen were lost in seven days, and on the last day there was a negative balance of 20.8 gm. In the third case, with a very low food intake, the urinary nitrogen averaged 21 gm. during the last seven days, and on one day the nitrogen excretion in the urine rose to 27 gm.

A similarly marked loss was evidently present in Ebstein's case, in which over 62 gm. of urea were found in the twenty-four hours' urine, and a number of other authors have seen much the same state of affairs. It is of interest that in Wey's case of chronic leukæmia, with a terminal very acute stage, the patient passed per day as much as 29 gm. of nitrogen in the urine. This violent disturbance is apparently not an essential part of the pathology of acute leukæmia, as there are one or two reliable cases on record that showed no marked loss; nevertheless, it appears from the observations that have been made to be a very pronounced and common feature.

Since leukæmia is a disease chiefly of the lymphatic glands and bone-marrow, it is but natural that the metabolism of the nucleins should show most striking evidence of disturbance. The excretion of uric acid and phosphates has been found very excessive in most cases, and some of the figures obtained by Magnus-Levy were exceedingly remarkable. One of his cases showed a loss of about 15 gm.

of P_2O_5 in fifteen hours, a most astonishing figure; and in the same case the uric acid excretion reached 12.22 gm., which, considering this patient's low food intake, was certainly twenty times the normal amount, while the purin bases were 0.321 gm. Similar, though less striking, losses have been observed by others. Wende, for example, reported a case in which the uric acid excretion reached nearly 5 gm. There are, however, a few cases on record in which the uric acid excretion was very slightly, if at all, increased.

It is a matter of great clinical interest, as well as pathological, to attempt to determine the cause of this profound disturbance of metabolism. Magnus-Levy, because of his own observations on nitrogen metabolism in hemorrhagic disorders, particularly in a case of purpura hemorrhagica, and because of a few observations of other investigators on the relation of hemorrhage to destructive changes in metabolism, reaches the conclusion that a large part, at any rate, of the tissue destruction in acute leukæmia is probably dependent upon the hemorrhages that occur in this disease. This seems to me to be very questionable. Magnus-Levy's work, and that to which he refers, has shown to be sure that hemorrhagic conditions are often associated with a marked nitrogen loss, and it is not improbable that a small amount of tissue destruction is dependent upon the hemorrhage itself. The experimental work of Hawk and Gies, for example, in which the only abnormality present was loss of blood, shows that mere hemorrhage does produce some tissue destruction. But in such experimental work, in which other abnormalities are not present, hemorrhage seems to cause but slight disorder of general metabolism, and hence it would appear to be an insufficient explanation of the extremely unusual conditions found in acute leukæmia. Furthermore, there are many cases of extensive purpura in which, even in the absence of accurate chemical investigations, the clinical appearances are such as to indicate pretty clearly that there cannot be in all cases a marked tissue loss. Therefore, since very severe abnormalities, besides the hemorrhage, are present in acute leukæmia, it seems to me that hemorrhage offers a very incomplete explanation of the metabolic disturbance in this disease. Violent acute disturbance of metabolism is usually due to some form of toxæmia, and it would appear more rational to look to such a cause in acute leukæmia than to attribute the major portion of the disturbance to hemorrhage; in other words, I should be prepared to learn that the hemorrhage and the disorder of metabolism were due to a common cause, probably a toxæmia.

The conditions in the case of purpura hemorrhagica that I shall at once mention indicate very strongly, I think, that while some tissue destruction occurred in this case also, hemorrhage was not the cause of it; and hence I believe that this case goes far to remove the basis of Magnus-Levy's hypothesis that hemorrhage is the main cause of the tissue destruction in acute leukæmia.

This patient was a boy aged sixteen years; he was admitted to the University Hospital on Dr. Musser's service December 15, 1904. Admission No. 3531. His trouble had begun eleven days before, with moderate joint pains, which had continued, but had not been severe at any time. There had been little swelling of the joints. Nine days before he was admitted to the hospital hemorrhagic spots had appeared in his skin; these had rapidly increased two days before his admission, and he had also had nausea and vomiting and cramping abdominal pain. The symptoms had become still worse on the following day. When admitted he looked very ill. He had nausea, frequent vomiting, and bloody bowel movements, and showed extensive subcutaneous hemorrhages, particularly on the arms and back. The symptoms continued severe, and on the 20th he was greatly prostrated and delirious, and his hemorrhages had increased to a most striking extent. I have never seen more widespread subcutaneous hemorrhages; they involved almost all the surface of the back, the arms, and much of the forearms; there were large extravasations in the skin over the front of the chest and abdomen, many large patches on the legs and a number on the face. There were also small hemorrhages into the conjunctiva; he had hemorrhages from the mouth and nose; he vomited some blood, and had also passed blood from the bowel and with the urine. I began with this day (*i. e.*, urine of 20th and 21st, marked 21st in table) observations of his excretion of nitrogen, phosphates, and uric acid. The excretion on the first two days represents total loss, as the patient was taking no food at this time; he was, indeed, losing more than is indicated by the urine, as he was vomiting almost constantly. It was also impossible on the 27th and 28th to estimate the intake accurately, as he had severe vomiting much of the time.

It should be noted that on December 21st he was still having hemorrhages, was vomiting violently, and was generally prostrated; on the 22d he was still very ill, but his gastric symptoms had, temporarily, largely subsided, and hemorrhage had, so far as could be determined, stopped entirely; on the 23d nearly all marked symptoms had ceased, and on the 24th he felt so well that I temporarily stopped my observations, as his attack seemed to be over. The point of chief interest is, however, that on the evening of the 24th he had a return of his gastric symptoms, and on the 26th the joint pains, abdominal pain, gastric symptoms, and prostration returned, but he had no discoverable return of hemorrhage. Observations of metabolism were again begun on this day. These symptoms lasted two days, and then decreased. Subsequently, though he had the signs of an acute nephritis, he improved with great rapidity in his general condition, and gained eleven pounds in a week. The figures for January 3d and 4th, therefore, represent his condition during convalescence. At this time he was getting between 11 and

12 gm. of nitrogen in his food. In the preceding periods, when he took food at all, he was probably getting at most 3 or 4 gm. of nitrogen.

Date.	Urine.	Nitrogen.	Uric acid.	P_2O_5.
December 21	1800	22.478	0.196	6.192
" 22	1700	23.738	Lost	4.012
" 23	1350	18.295	0.178	3.481
" 24	870	14.323	0.158	1.531
Return of symptoms, but no hemorrhage:				
December 27	1560	22.579		2.280
" 28	1470	20.580		1.705
Convalescent:				
January 3	850	6.718		0.950
" 4	1275	6.612		0.918

For the first two days these figures show, as far as the nitrogen and P_2O_5 are concerned, conditions that are very similar to those in my case of acute leukæmia, and in the majority of the other cases in which metabolism has been observed, namely, a profound loss. The next day, when there was no hemorrhage and the symptoms were all disappearing, there was a very marked decrease in the nitrogen and P_2O_5 loss; while on the fourth day the conditions were approaching the normal. The first four days, therefore, apparently support Magnus-Lævy's view that the hemorrhage may be responsible for much of the metabolic disturbance. The conditions on the 27th and 28th, however, indicate directly the contrary. At this period hemorrhage was absent, but the renewed metabolic disturbance was only slightly less severe than that at the time when hemorrhage was occurring in wide areas, from the mucous membranes and into the skin. The conditions on January 3d and 4th are those to be expected in a convalescent; the man was, as his weight indicated, showing a marked retention, and he was evidently using this in building up tissue.

One case does not, of course, prove or disprove an hypothesis. Nevertheless, the conditions in this case; the fact that extensive hemorrhage often occurs without the clinical appearance of tissue loss; the knowledge that hemorrhage does not necessarily occur in acute leukæmia; and the fact that, besides the hemorrhages, evidences of grave disorders of other kinds are present in acute leukæmia and in many other hemorrhagic cases: all these unite, I think, to make it appear extremely improbable that the tissue loss should be attributed in considerable extent to the hemorrhage, and indicate, on the contrary, that the cause of both hemorrhage and tissue loss must be sought together in the nature of the disturbance that produces the disease.

At the time that I observed my case of acute leukæmia I suggested that it is probable that the tremendous tissue loss is due to some

pathological disturbance of autolytic processes. This suggestion was at that time almost a pure hypothesis, but the hypothesis was based upon the fact that similar metabolic conditions are seen in pneumonia at the time of the crisis, and in acute yellow atrophy of the liver; and in these diseases sudden and very extensive autolytic tissue destruction occurs; in the lung exudate in the one case, in the liver in the other. Since that time further experiment has led physiologists and pathologists to a general acceptance of the importance of autolysis in a great number of physiological and pathological conditions, and the suggestion that the metabolic disturbance in acute leukæmia is a disturbance of autolysis is on general grounds now better justified.

More direct evidence in favor of this view is found, I think, in observations that I have recently made on the influence of the *x*-ray on metabolism in chronic leukæmia.[1] In a case successfully treated there was, immediately upon the use of the *x*-ray, a most astonishing increase in the excretion of nitrogen, uric acid, purin bases, and P_2O_5. The excretion was, on the whole, fully doubled and that of uric acid, purin bases, and P_2O_5 was ultimately much more than doubled. On the other hand, a case of chronic leukæmia that was unsuccessfully treated with the *x*-ray showed almost no change in metabolism as a result of this treatment, and soon died, with constantly decreasing excretion. The fact that normal persons respond to *x*-rays by increase of metabolism, and that the leukæmic successfully treated showed even more striking response, while, on the other hand, the leukæmic unsuccessfully treated, and also two persons with gout had no noteworthy alterations in metabolism as a result of *x*-ray exposures, led to the conclusion that the changes in metabolism that occurred as a result of exposure to *x*-rays are due to some response in the individual and not to any direct physical or other effect of the *x*-ray upon the tissues. The great rapidity of action of the *x*-ray upon metabolic processes, together with the other facts already mentioned, makes it seem highly probable that *x*-rays act in some way in increasing the ferment-like metabolic processes that are normal in the body; in other words, in stimulating autolysis. This becomes almost a definite fact when one considers that Neuberg has shown experimentally that radium increases autolysis *in vitro*.

The conditions in the case of chronic leukæmia that was successfully treated with *x*-rays bore a striking resemblance to the conditions in acute leukæmia, though they were not so extremely pronounced. The fact that autolytic processes can apparently be thus—*i. e.*, under the influence of the *x*-ray—changed from their normal state to one in which there is very unduly rapid tissue

[1] Reported, with Dr. Musser, in the University of Pennsylvania Medical Bulletin, September, 1905.

destruction suggests strongly, I think, that the conditions in acute leukæmia are actually due to some cause that similarly, though more markedly, excites an increase of autolytic tissue destruction.

Until the observations on the influence of the æ-ray on metabolism in leukæmia were made I was naturally inclined to believe that the conditions in acute leukæmia are a part of the malignancy of the disease. The effect of the æ-ray on chronic leukæmia, however, suggests strongly that the tissue destruction in acute leukæmia may be not an essentially malignant process, but one that is of benign purpose. In chronic leukæmia æ-ray exposures produce, in cases successfully treated, a remarkably rapid tissue destruction that is evidently, from the clinical course of these cases, benign in its influence upon the organism. It seems wholly possible, therefore, that the destruction that occurs in acute leukæmia is a response of the organism to the leukæmic tissue hyperplasia, and is an attempt to control this and to destroy the leukæmic tissue. It is not improbable, however, that if this is the case metabolic processes run amuck in the destruction of leukæmic tissue, and carry the destruction so far that other tissues are involved to a dangerous degree; and, too, death is probably often hastened by the intoxication that results from the products of the rapid tissue destruction.

The fact that tissue destruction has not been found greatly increased in all cases of acute leukæmia, but is so in most instances, tends rather to emphasize than to disprove this suggestion that the tissue destruction is an attempt at reaction on the part of the diseased individual, and that the purpose of the reaction is the destruction of diseased tissue. The fact that it is not constant indicates that the severity of the disease is not chiefly dependent upon the tissue destruction, and this latter point may readily be brought in harmony with the view that the tissue destruction is a part of the fight of the organism against the disease. Considering, again, the analogy between acute leukæmia and chronic leukæmia, when the latter is being treated by æ-rays, the above suggestion concerning the purpose of the tissue destruction in acute leukæmia becomes more emphatic when we remember that the tissue destruction in chronic leukæmia that results from æ-ray treatment is limited almost entirely, perhaps entirely, to the lymphatic tissues and bone-marrow—that is, to the diseased tissues.

In conclusion, I would refer to the curiously low excretion of uric acid in the case of purpura hemorrhagica, which is of interest when contrasted with the large loss of nitrogen, and particularly with the extremely large amount of P_2O_5 lost. As will be noticed, on the day when hemorrhage was continuing the P_2O_5 excretion was above 6 gm., and on the next day it was still about 4 gm.; amounts that are very excessive at best, and that are particularly so in this instance, because there was no intake of P_2O_5.

Indeed, a noteworthy amount more than this was probably excreted through his vomit. On the other hand, the uric acid figures are about normal for a person who is temporarily taking no food; in fact, they are even rather low for such circumstances. The sources of the P_2O_5 are chiefly bone, nucleins, and other parts of tissues, such as lecithin, that contain much phosphorus. That it came in this instance largely from the bones is, of course, very improbable at best, and is practically ruled out by the fact that the general nitrogen excretion was so great that it indicates marked destruction of nitrogenous tissue. It is, of course, possible that lecithin destruction, which, like bone destruction, would yield no uric acid, was responsible for the excessive P_2O_5 excretion, but it is very difficult to conceive of a process that would cause such an extensive destruction of lecithin and of nitrogenous tissues in general and leave the nucleins uninvolved. It seems far more probable that, as is usually the case, it came from nuclein, and that uric acid had been produced; but that, owing to some peculiarity in the disease or the individual, in this case the uric acid had been destroyed and was probably excreted as urea; while in most of the acute leukæmia cases that have been studied the uric acid was not further changed, but was excreted as such. The observations of Magnus-Levy and others on hemorrhagic diseases did not include estimations of the uric acid and P_2O_5, so that I cannot state whether the conditions that I found are frequent in purpura and other hemorrhagic conditions, or were merely peculiarities of this individual case. It would be of interest to determine whether this is a common feature in purpura hemorrhagica as contrasted with other hemorrhagic conditions. If so, it would show that the metabolic disturbances of acute leukæmia and purpura hemorrhagica, though they may closely resemble each other in a gross manner, may exhibit some striking differences in details. It would then be further evidence of the fact that has long been so evident, both clinically and pathologically, that hemorrhagic conditions of various sorts are very different in their natures, though the superficial resemblance provided by the hemorrhages leads, in a clinical discussion, to the inclusion of a number of those that are least understood under one general heading.

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SOME OBSERVATIONS ON THE EFFECT OF CERTAIN DIET CURES IN DIABETES MELLITUS.

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"In no other disease is the diet of greater importance than in diabetes. It may be said that there is no rational treatment for diabetes other than the dietetic. When the physician and patient are wearied and discouraged by the great duration of this disease they seek recourse to water cures and bathing resorts, as well as aid from drugs, old and new, of animal, vegetable, or mineral origin. Such measures can only be followed as side issues, and even then can only be practised with great danger, even for a short period of time; for in diet alone do we possess the actual remedy which fulfils the indications for treatment, namely, the maintenance of nutrition, the increase in the ability of the body to assimilate sugar and the avoidance of complications" (von Noorden).

It is not our intention to attempt to cover the field of the dietetic treatment of diabetes in this paper. We wish, however, to bring forward certain diet cures which have assumed some importance during the past few years. These are three in number: the milk cure, the potato cure, and the oatmeal cure.

Milk Cure. According to Naunyn,[1] the merits of a strict milk diet in certain cases of diabetes have been recognized for some years. Rollo advised its use in quantities of a litre a day, and Donkin[2] considered it a valuable food in the treatment of this disease, believing in its efficacy in many cases in reducing and even causing the sugar to disappear entirely from the urine. Winternitz and Strasser[3] and Kolisch[4] have more recently attempted to emphasize the truth of this observation, believing that a strict milk diet will either diminish or even cause the glycosuria to disappear entirely in from twenty-four to forty-eight hours in most cases of this disease. A certain number of patients are entirely relieved by means of the milk cure and remain so after again being placed on a diet containing a considerable amount of carbohydrate material. Naunyn points out that even in severe forms of diabetes ½ a litre of milk a day is often well borne without increasing the glycosuria. He observes, however, that diabetic patients react differently to milk-sugar, and that a trial must, therefore, be made in each individual case. He also draws attention to the fact that often the

unfavorable effect of milk can be detected only after a lengthy and continuous use of this article of food.

A. Berger[5] also studied the effect of a strict milk diet in diabetic glycosuria. He showed that a patient who was entirely free from sugar in the urine on taking 50 grams of bread and ¼ of a litre of milk a day, passed 3 grams of sugar after taking 3 litres of milk in the same length of time. Another diabetic patient who excreted 64 grams of sugar after taking four rolls and 2 litres of milk, produced but a few grams of sugar on a strict milk diet of 2 to 3 litres. Hirschfeld,[6] Blumenthal,[7] Lenné,[8] and von Noorden[9] all believe that it is impossible to formulate any definite rules regarding the effect of milk in diabetes, but consider that every case must be tested individually to determine the tolerance for this particular article of food.

According to von Noorden, milk should be never given in mild forms of diabetes in which glycosuria appears on the taking of this food. Von Noorden also admonishes against the use of a strict milk cure in all cases of diabetes, even when glycosuria does not appear after taking as much as 4 litres of milk daily, as this form of food frequently produces an intense anorexia which is often very difficult to overcome. He advises that in those cases of diabetes in which glycosuria is absent on a milk diet to vary the milk with kefir, buttermilk, kumyss, and cream. In order to avoid any deleterious effect of the milk-sugar, von Noorden recommends the use of the so-called diabetic milk (Gärtner's process), from which a large portion of the milk-sugar has been removed. This product contains but 1 per cent. of milk-sugar.

Hutchison[10] advises the following method for preparing diabetic milk, which contains but a trace of sugar:

Sugar-free Milk for Diabetic Feeding. Take 1 litre of skim milk, heat to a temperature of 30° C., and add 10 c.c. of glacial acetic acid, diluted with 100 c.c. of water. Mix and allow the mixture to stand for about fifteen minutes. Collect the separated casein, and let it drain on very fine muslin, using no pressure. Remove the casein to a mortar, rub into a smooth paste, add ½ litre of distilled water, and strain as before. Repeat this washing of the casein twice. Transfer to a mortar, rub until quite smooth, and add 2½ grams of potassium hydrate dissolved in 100 c.c. of water (or as much of the potassium hydrate as is necessary to make the product just alkaline to phenolphthalein). Add 100 grams of ordinary Devonshire clotted cream, 5 grams of gelatin previously dissolved, 0.06 grams (1 gr.) of saccharine and water, at about 38° C., up to 1 litre. Lastly, strain through fine muslin.

Our own observations with the use of milk as a form of diet in diabetes are in accord with those of von Noorden and other recent writers. We have rarely found it advisable to place any of our patients on an exclusive milk diet, except in those severe cases

of diabetes in which diacetic acid is present in the urine and in which the patient is threatened with the onset of diabetic coma or in which this condition had already set in. On the other hand, we have utilized from ½ to a litre of milk daily in many of our cases of diabetes in addition to other allowable foods, often with excellent results. It is important, however, as has already been pointed out, to observe the effects of this form of food, lest untoward effects be produced and great injury be done the patient. The following cases illustrate the effect of a milk diet in diabetic cases:

CASE I.—A. B., a male, aged fifty-eight years; the patient has had diabetes for about a year; he has lost ten pounds in weight. On an unrestricted diet he passed as much as 3.9 per cent. of sugar in his urine (116 grams daily). When placed on a diet free of carbohydrates (von Noorden's standard diet) his urine became free of sugar; which was still the case when as much as 150 grams of bread were added to this diet. This case can therefore be considered a mild form of the disease. This patient could consume a litre of milk a day without producing glycosuria.

On the other hand, Case II., J. T., female, aged forty-four years, with conditions almost identical with Case I., showed a far less degree of tolerance for milk. This patient passed 2.8 of sugar (68 grams per day) on an unrestricted diet; and no sugar, either on a von Noorden's standard diet or on this diet, with the addition of 150 grams of bread; yet on the addition of ½ litre of milk, 1.8 per cent. of sugar (40 grams per day) was eliminated, and on 1 litre, 2.5 per cent. (58 grams).

CASE III.—E. K., a male, aged forty-two years, illustrates the effect of milk in a severe form of diabetes. This patient had lost forty-four pounds in flesh, and on an unrestricted diet passed 4.8 per cent. of sugar (148 grams daily); on a von Noorden's standard diet, 3.2 per cent. of sugar (83 grams daily). His urine, even under these conditions, presented considerable quantities of diacetic acid. When placed upon an exclusive milk diet for ten days, the percentage of sugar decreased to 2.1 per cent. (42 grams daily), the diacetic acid disappeared, and he began to increase slightly in flesh.

CASE I.—A. B., male, aged fifty-eight years; duration of disease one year; former weight 148 pounds.

Observation.	Diet per day.	Daily quantity of urine c.c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Present weight.
1	Unlimited . . .	2980	1037	3.9	116	None	138 lbs.
2	Standard diet (von Noorden)	2010	1026	None	None	"	
3	Standard diet + 150 gm. bread	2020	1028	"	"	"	
4	Standard diet + 150 gm. bread + 1 litre milk	2000	1028	"	"	"	

CASE II.—J. T., female, aged forty-four years; duration of disease six months; former weight 122 pounds.

Observation.	Diet per day.	Daily quantity of urine c.c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Present weight.
1	Unlimited diet . .	2450	1032	2.8	68	None	116 lbs.
2	Standard diet . .	2000	1026	None	None	"	
3	Standard diet + 150 gm. bread	2000	1021	"	"	"	
4	Standard diet + 150 gm. bread + ½ litre milk	2280	1039	1.8	40	"	
5	Standard diet + 150 gm. bread + 1 litre milk	2350	1030	2.5	58	"	

CASE III.—E. K., male, aged forty-two years; duration of disease three years; former weight 188 pounds.

Observation.	Diet per day.	Daily quantity of urine c.c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Present weight.
1	Unlimited diet . .	3100	1040	4.8	148	Present	144 lbs.
2	Standard diet . .	2600	1036	3.2	83	"	
3	Milk diet (3 litres) .	2000	1030	2.1	42	None	

POTATO CURE.—Mossé[11] first advised the potato cure as a means of treating patients suffering with diabetes. His plan is to replace all carbohydrates by potatoes for a period of several weeks. Patients are to receive 1500 grams of potatoes, replacing 500 grams of wheat bread in as much as according to his experiments patients could tolerate from two and one-half to three times as much of potatoes (weighed raw) as they could of wheat bread. Potatoes are relatively poor in carbohydrates containing from 16 to 22 per cent. of starch, while wheat bread contains about 60 per cent. During the time of the potato treatment no other carbohydrates are to be consumed. Mossé found a marked diminution in the glycosuria and polyuria while the patient was taking the potato starch, as well as a diminution in thirst and an increase in strength. He also observed that a certain degree of tolerance for wheat bread was produced after the patient had been on the potato cure for several days. He attributes the beneficial effect of this diet to the large proportion of water and potassium salts contained in the potato. The large quantity of fluid diminishes the thirst, while the potassium salt appears to improve the general nutrition. In 23 diabetic patients to whom diets of bread and potato were alternately given, an advantage in favor of the bread was found in only 1 case.

Offer[12] has confirmed the observation of Mossé, and von Noorden,[13] too, extols this plan of treatment, and recommends that the potato need not only be given as the only carbohydrate food in

diabetes, but also that it may be utilized to replace a certain portion of the bread; for instance, in a diet in which 75 to 125 grams of wheat bread are allowable an amount of potato represented by a third of this quantity can be substituted for it; so that in this instance 75 to 125 grams of potato can be consumed daily, leaving as much as 50 to 85 grams of bread still to be eaten. In this way the variety of carbohydrate food is increased, and at the same time large quantities of fat can be consumed in the form of butter, which may be added to the potatoes. This is a matter of great importance for inasmuch as a large proportion of the carbohydrates are excluded in this disease, this heat-producing food which is eliminated must be substituted for by some other form of food, and this is best accomplished by means of fats, such as butter.

In our own observations with the use of the potato cure in the treatment of diabetes, we have never followed Mossé's plan, that is, by replacing all carbohydrates by potatoes, but have rather followed the method suggested by von Noorden, replacing a portion of the bread by this form of food. A patient who is permitted to consume 100 grams of bread a day can take 300 grams of potatoes, so that at least a certain proportion of the bread can be replaced by this form of food. If care be taken to regulate these proportions we have never seen the slightest harmful effect produced by the use of potatoes, even in severe forms of diabetes. In a not inconsiderable number of cases very beneficial results were obtained by us. The potato has an additional advantage, in that it lends variety in furnishing carbohydrate food in as much as it can be prepared in various ways, baked, boiled, fried, mashed, etc., and again as being a means, as has already been pointed out, through which fatty food, such as butter, can be furnished the body in rather large quantities. The following cases illustrate the effect of a potato diet in various forms of diabetes:

CASE IV.—J. H., a male, aged forty-nine years, who had suffered with diabetes for nine months and who had lost twenty-two pounds in weight, illustrates how the potato diet acts in a mild form of diabetes. This patient who passed 4.1 per cent. of sugar in his urine (115 grams daily) on an unrestricted diet was free of all sugar in his urine on a standard diet, and also when 150 grams of bread were added to the diet; the absence of sugar continued when to this diet 50 grams of bread and 300 grams of potatoes were added. This patient passed about 1 per cent. of sugar (20 grams daily) on the usual diabetic diet, with the addition of 60 grams of bread, but when only 20 grams of bread were allowed and 120 grams of potatoes he passed but 0.9 per cent. of sugar in his urine (18 grams daily).

CASE V. illustrates how the potato diet acts in a moderately severe form of diabetes. This patient, a female, aged sixty-two years, had had diabetes for two years; she had lost twenty-eight pounds in weight; on an unlimited diet she passed 4.6 per cent. of sugar (138 grams daily; on a standard diet, none; on a standard diet,

with the addition of 60 grams of bread, 2.4 per cent. (67 grams daily); on a standard diet, with 20 grams of bread and 120 grams of potatoes, but 2.1 per cent. (54 grams daily); on the usual diabetic diet, with the addition of 60 grams of bread, 3.2 per cent. (83 grams daily); while on the same diet, with but 20 grams of bread and 120 grams of potatoes, but 2.8 per cent. (73 grams daily).

CASE VI. illustrates the effect of the potato diet on a severe form of diabetes. This patient was a female, aged thirty-seven years, who had been suffering with the disease for three years.

On an unlimited diet the patient passed as much as 5.1 per cent. of sugar (163 grams daily); on a von Noorden standard diet this amount was reduced to 3.6 per cent. (90 grams daily); when to this standard diet 60 grams of bread were added, the patient passed 4.5 per cent. of sugar (135 grams daily), which was reduced to 4 per cent. (112 grams daily); when one-third of the bread was replaced by a proportionate amount of potato. On the usual diabetic diet containing 60 grams of bread, this patient passed 4.8 per cent. (144 grams daily) of sugar, which was reduced to 4.6 per cent. (133 grams daily), when 40 grams of the bread were replaced by 120 grams of potato.

CASE IV.—J. H., male, aged forty-nine years; duration of disease nine months; former weight 180 pounds.

Observation	Diet per day	Daily quantity of urine c c	Specific gravity	Percent of sugar	Total quantity of sugar per day	Diacetic acid	Present weight
1	Unlimited diet . .	2800	1034	4 1	115	None	162 lbs
2	Standard diet	2050	1026	None	None	"	
3	Standard diet + 150 gm bread	2120	1026	"	"	"	
4	Standard diet + 50 gm bread + 30 gm potato	2020	1028	"	"	"	
5	Usual diabetic diet + 60 gm bread	2140	1028	1 0	20	"	
6	Usual diabetic diet + 20 gm bread + 120 gm potato	2020	1024	0 9	18	"	

CASE V.—Z. L., female, aged sixty-two years; duration of disease two years; former weight 162 pounds.

Observation	Diet per day	Daily quantity of urine c c	Specific gravity	Percent of sugar	Total quantity of sugar per day	Diacetic acid	Present weight
1	Unlimited diet .	3000	1038	4 6	138	None	134 lbs
2	Standard diet	2060	1024	None	None	"	
3	Standard diet + 60 gm bread	2800	1032	2.4	67	"	
4	Standard diet + 20 gm bread + 120 gm potato	2600	1030	2 1	54	"	
5	Usual diabetic diet + 60 gm bread	2620	1032	3 2	83	"	
6	Usual diabetic diet + 20 gm bread + 120 gm potato	2610	1032	2 8	73	"	

CASE VI.—M. N., female, aged thirty-seven years; duration of disease three years; former weight 172 pounds.

Observation.	Diet per day.	Daily quantity of urine c.c.	Specific gravity.	Percent of sugar.	Total quantity of sugar per day	Diacetic acid.	Present weight.
1	Unlimited diet . .	3200	1040	5.1	163	None	139 lbs.
2	Standard diet . .	2500	1034	3.6	90	"	
3	Standard diet + 60 gm. bread	3000	1036	4.5	135	"	
4	Standard diet + 20 gm. bread + 120 gm. potato	2800	1035	4.0	112	"	
5	Usual diabetic diet + 60 gm. bread	3000	1038	4.8	144	"	
6	Usual diabetic diet 20 gm. bread 120 gm. potato	2900	1036	4.6	133	"	

OATMEAL CURE. Von Noorden[14] first pointed out that certain patients suffering with diabetes, who, notwithstanding the fact of being on a strict diet, continued to eliminate sugar, could at times be quickly and permanently relieved of this condition by being placed upon a diet of large quantities of oatmeal. He also pointed out that in many severe forms of diabetes oatmeal is twice as well borne as an equivalent quantity of bread, and frequently by gradually increasing the quantity of oatmeal the glycosuria did not increase, while acetone and diacetic acid disappeared. The beneficial results were, however, only obtained when the oatmeal represented the largest proportion of food consumed, when no other carbohydrates were taken and no meat eaten. Von Noorden advises that the oatmeal cure should be undertaken for eight to fourteen days only, inasmuch as the patient will become satiated with this article of food after this length of time. In some instances, after a return to the usual diet, the tolerance for carbohydrates increases, while in others it does not, and glycosuria rapidly returns, and frequently diacetic acid, too.

In a more recent publication von Noorden[15] reports the results of his observations in 100 cases of diabetes treated by means of the oatmeal diet.

The results are classified into five classes:

1. Those cases in which it was impossible to produce a glycosuria, notwithstanding a strict diet. In these instances remarkable results were frequently obtained by means of the oatmeal cure, which could not have been obtained in any other way. It was often possible in these cases to allow a certain proportion of carbohydrates in addition to the usual diabetic diet without producing glycosuria.

2. In another class of cases good results were obtained from the oatmeal cure, the results were not permanent, although by means of this form of treatment the onset of diabetic coma was prevented.

3. In another class the tolerance for carbohydrates did not increase

after inaugurating the oatmeal cure, but aceton diminished and remained at a minimum, even after a return to the usual diet.

4. In not an inconsiderable proportion of cases the glycosuria increased rather than diminished on the oatmeal diet.

5. In mild forms of diabetes without diaceturia the oatmeal diet was badly borne.

In a general way it may be stated that the oatmeal cure is especially useful in those forms of diabetes exhibiting diacetic acid in the urine and that in mild forms it is not only a useless form of treatment, but may even prove harmful. On the other hand, as von Noorden points out, each case of diabetes should be studied individually and daily examinations of the urine should be made, in order to determine whether the special case is being benefited by this form of treatment. In a recent communication, Mohr,[10] working under von Noorden's direction, shows that the oatmeal cure is especially useful as a means of diminishing the acetone in diabetic cases.

In diabetic coma von Noorden recommends the oatmeal cure as preferable to the milk cure, inasmuch as it usually diminishes the aceton bodies more quickly. He believes the beneficial effect of the oatmeal is due to the substances contained therein, especially the salts; by analysis of the feces he showed that the lessened excretion of sugar in the urine was not due to a lessened absorption of the oatmeal in the intestines.

Von Noorden's directions for preparing the oatmeal are as follows: Knorr's oatmeal or Hohenlohe's Haferflocken (but any good oatmeal may be used) is well cooked for a considerable length of time in water with the addition of a bit of salt; while cooking butter and a vegetable albumin are added or the white of egg beaten and strained may be mixed with it, as the oatmeal cools. Roborat or a rice albumin (Bremer Brotfabrik) is recommended as being especially palatable mixed with the oatmeal. The quantities to be taken daily are: oatmeal, 250 grams; albumin, 100 grams; butter, 300 grams.

The meals are taken at two-hour intervals; in addition, von Noorden permits cognac or wine and some strong black coffee; after a few days five to seven eggs, but no meat; after eight to fourteen days a vegetable diet day is interposed and occasionally at rare intervals, if the oatmeal diet is to be kept up for any length of time, a small quantity of meat or fish is allowed to relieve the monotony of the diet and render it bearable. The return to the ordinary diet must be made cautiously, lest the acetone bodies increase to an alarming degree.

Our results with the oatmeal diet have been very satisfactory. In mild forms of diabetes this treatment should never be utilized. This is well illustrated in Case VII. The patient, a man, aged sixty-two years, had suffered with diabetes for six months; on an

unrestricted diet he passed 2.2 per cent. of sugar in his urine (48 grams daily); on a standard diet the urine became sugar-free, which was also the case when 150 grams of bread were added to the standard diet; on an oatmeal diet the patient passed as much as 4 per cent. of sugar (112 grams daily), which was reduced to 1 per cent. (24 grams daily) when the patient was placed on the usual diabetic diet.

The oatmeal cure has its place only in the severe forms of diabetes. In these cases we have obtained remarkable results. In certain cases it is possible to rid the urine of sugar entirely when this is impossible on an entirely carbohydrate-free diet (standard diet).

Case VIII. illustrates this condition. This patient, a female, aged forty-four years, had had diabetes for eight years; on an unrestricted diet she passed 5.8 per cent. of sugar (185 grams daily), which was reduced to 3.1 per cent. (86 grams daily) on a standard carbohydrate-free diet. This patient was placed on the oatmeal diet for eight days, and the sugar disappeared entirely from the urine; it reappeared when the patient was placed on the usual diabetic diet, but never reached such large proportions as it did originally.

In another class of cases the oatmeal diet not only occasions a disappearance of the sugar in the urine, but also the diacetic acid. The condition is represented in Case IX., F. F., a female, aged fifty-nine years, who had had diabetes for four years; on an unrestricted diet she passed 6.8 per cent. of sugar (250 grams daily); on the usual diabetic diet 5.4 per cent. (180 grams daily); after being placed on an oatmeal diet for eight days both sugar and diacetic acid disappeared. While sugar reappeared in the urine after the patient had been placed on the usual diabetic diet, it did not reach the large percentage it had gotten to before, nor did diacetic acid reappear.

Finally, there are a number of cases, of which Case X. is a representative, in which, while the percentage of sugar present in the urine is not materially affected by the oatmeal cure, diacetic acid disappears under its influence and remains absent. Thus, Case X., K. C., female, aged thirty-eight years, who had had diabetes for two years, passed 6.2 per cent. of sugar (248 grams daily) when taking an unrestricted diet; on the usual diabetic diet, this amount was reduced to 5.5 per cent. (176 grams daily). The percentage of sugar remained at 5 per cent. (170 grams daily), even after the oatmeal cure had been inaugurated; but whereas diacetic acid had been present while this patient had been on an unrestricted diet, as well as a diabetic diet, it disappeared entirely after the patient had been taking the oatmeal for a few days.

In conclusion, we believe we are justified in asserting that the milk cure, potato cure, and oatmeal cure are valuable aids in the treatment of diabetes.

Great care, however, must be practised with the use of these various forms of diet. Each case must be studied individually and must be carefully watched in order to obtain the very best results.

CASE VII.—J. L., male, aged sixty-two years; duration of disease six months; former weight 186 pounds.

Observation.	Diet per day.	Daily quantity of urine c c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Weight.
1	Unlimited diet . .	2200	1028	2 2	48	None	175 lbs.
2	Standard diet . .	1800	1026	None	None	"	
3	Standard diet + 150 gm. bread	2000	1026	"	"	"	
4	Oatmeal diet . .	2800	1038	4.0	112	"	
5	Ordinary diabetic diet	2400	1026	1.0	24	"	

CASE VIII.—T. S., female, aged forty-four years; duration of disease eight years: former weight 172 pounds.

Observation	Diet per day.	Daily quantity of urine c c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Weight.
1	Unlimited diet . .	3200	1040	5.8	185	None	124 lbs.
2	Standard diet . .	2800	1032	3.1	86	"	
3	Oatmeal diet . .	2000	1024	None	None	"	

CASE IX.—F. F., female, aged fifty-nine years; duration of disease four years; former weight 184 pounds.

Observation.	Diet per day.	Daily quantity of urine c c.	Specific gravity	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid	Weight.
1	Unlimited diet . .	3800	1042	6.8	250	Marked	140 lbs.
2	Ordinary diabetic diet	3400	1040	5.4	180	"	
3	Oatmeal diet .	2400	1028	None	None	None	

CASE X.—K. C., female, aged thirty-eight years; duration of disease two years; original weight 130 pounds.

Observation	Diet per day.	Daily quantity of urine c.c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Weight.
1	Unlimited diet .	4000	1040	6 2	248	Marked	110 lbs.
2	Ordinary diabetic diet	3200	1038	5 5	176	"	
3	Oatmeal diet . .	3400	1036	5 0	170	None	

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GANGRENE OF THE TONSIL.[1]

BY CHARLES W. RICHARDSON, M.D.,
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AT the twenty-fifth annual meeting of the American Laryngological Association, held in Washington, I had the pleasure of presenting a case of this unusual condition, which was read by title. Since the report of this case, I have had the fortune to observe another case of a similar character, and, as the condition seems to be of such unusual character, I have incorporated my first case in this paper, as well as given abstracts of the only 3 cases which I find under the title of gangrene of the tonsil in medical literature. It seems passing strange that this condition should be so infrequently observed; more strange, if of such infrequent occurrence, that I should have had 2 cases under my observation within fourteen months. The diagnosis presents no difficulty; the physical signs and the constitutional manifestations are too positive. The exclusive bacteriological investigation and the rapid fatal termination can leave no doubt in the minds of the most skeptical diagnostician. The characteristic stench of the disorganized tissue is, like the odor of atrophic rhinitis, once recognized, never forgotten.

CASE I.—On March 1st I was called in consultation to see a patient residing about nine miles from Washington. It was late in the afternoon when I reached the patient's house, and it was nearly an hour later before the consultants were with me.

On the presentation of the case to me by one of the consultants and carefully going over the history and the temperature sheets, I

[1] Read before the American Laryngological Association, 1905.

could see at once that the case, whatever might be its cause, was one of extreme sepsis. The history as presented to me was as follows:

The patient was a man of robust physique, aged forty-five years, addicted to excesses. About ten days before he had returned from a trip to the North, at which time he had been using alcoholics to excess. When he first came under the observation of the physicians in charge, he had considerable difficulty in swallowing, marked infiltration of the right side of the neck, with other characteristic symptoms of quinsy. In a couple of days it was stated that the patient had a discharge of pus from the nose and mouth, with which there was a subsidence of the swelling in the right side of the neck, a restoration of the normal voice, no further difficulty in deglutition, and the return of the ability to open the mouth freely and widely. About two days thereafter the physician in charge saw what he supposed to be some white spots on the right tonsil. He then thought that the case was probably one of diphtheria, although there was a doubt in his own mind as to the diagnosis. The next day, however, he administered 3000 units of antitoxin. On the second day thereafter there were 3000 more units given. From the first administration until I saw him he received in all 16,000 units of antitoxin. Even under this repeated use of antitoxin there was no improvement in the patient's condition. He gradually but progressively grew worse. On the ninth day of his illness the patient became delirious, and on the tenth and last day of his life I was called in consultation.

At this time the patient's temperature was 102°, his pulse 120, soft and compressible; and he was in a muttering delirium. On inspection, which was comparatively easy for one in a delirium, and in such a serious condition, I was at first struck by the absence of the characteristic diphtheritic odor of the breath, and was impressed by the peculiar odor which is due to sloughing tissue. On depressing the tongue I was quite unprepared for the sight which I observed. On the right side the tonsil, the anterior and the posterior pillar, and as far down in the pharynx as I could see was one continuous mass of grayish, brownish, putty-like slough. The left tonsil and the curtain were perfectly normal. I particularly noticed the fact that there had already formed a complete line of demarcation between the necrotic and the normal tissue. The examination was prolonged until a careful and thorough inspection was made of the diseased area, and both the consultants had examined it with the same care. On consideration of the case, they both told me that the first they had noticed of the sloughing was the day before, when there was a narrow white line of diseased tissue; but, judging from the complete line of separation, I was rather inclined to believe that their examination was not as perfect a one as had been made by me. The patient was *in extremis*, and died two hours after I first saw him in consultation with Drs. Chappil and Kleinschmidt.

CASE II.—On May 17, 1904, I was called in consultation by Dr. Edward F. Pickford to see an unusual case of ulceration of the tonsil. The subject was a man, aged thirty-six years. His occupation was that of an insurance agent. He was married, and, as far as could be ascertained, led a moral life in every respect. Denied the existence or possibility of venereal infection; no past evidence of such infection. Personal habits good. So far as could be elicited, he had led a uniform, regular life, from which there had been no deviation in past six months. Family history good. Had had the usually infectious diseases of childhood. Dr. Pickford told me that he had been called to see the patient on May 4th. The patient stated to him that he had had a sore mouth and sore throat for several days. Mastication and deglutition were very painful and difficult. The patient was very fretful and extremely despondent. The temperature was 99.8°. On inspection the breath was noted to be extremely foul; the tongue heavily coated; the gums congested, swollen, bleeding readily, and separated from the teeth. There were some isolated grayish patches along the lower borders of the gums. The tonsils, particularly the right one, were very much enlarged and tender to the touch, at this time showing no evidences of ulceration. Two days later ulcerative patches were manifest on the right tonsil. By May 12th patient's condition had markedly improved. His temperature was normal; he could eat and drink with comfort, and his depression of spirits had given way to his usual happy demeanor. The swollen gums had receded, the patches on gums and tonsils had disappeared, and the tonsils were much reduced in size. Dr. Pickford discharged himself, but was recalled to the patient May 15th. On this latter visit Dr. Pickford found quite a change in the condition of his patient. The principal lesion was now in the tonsillar region. The left tonsil was covered with a dirty, grayish-looking patch. The mouth was nearly normal. The throat was extremely sore and the patient was markedly irritable and greatly depressed. Temperature, 99⅗°. As the deposit on the tongue looked diphtheritic, Dr. Pickford made a culture, with negative result. Dr. Pickford told me on our consultation that he thought the case at first to be one of ulcerative stomatitis of scorbutic origin. Examination made by Dr. Pickford of other systems and organs of the body showed them to be in apparent normal condition. When I first saw this patient his countenance showed an exceedingly anxious expression; he was very restive, but not unduly nervous. Complained greatly of difficult breathing, and was excessively despondent and anxious as to his welfare. Told me that his throat pained him greatly; that it was almost impossible to swallow food, and that he feared to assume a recumbent posture for fear that his throat would close. Temperature, 100⅕°. On examination, under excellent illumination, I found no change in the buccal cavity. There was a redness along the edges of the gums and they seemed

slightly raised from the teeth. On examination of the fauces the change in the right tonsil impressed me at once. The whole surface of the right tonsil was covered with a blackish scab-like exudate, which had an exceedingly offensive odor. There was no change in the left tonsil or in the other portions of the oral pharynx. The blackish exudate was subjected to a most vigorous curetting, nevertheless, without being able to reach healthy tissue. The tissue removed had all the appearances and smell of necrotic tissue. A bacteriological examination had already excluded the presence of the Klebs-Loeffler bacillus. The case was diagnosed as dry gangrene of the tonsil. On May 18th I again saw the patient. His general condition was slightly improved, his temperature the same, although the local condition looked more unfavorable. There was a recurrence of the slough all over the area from which it had been removed the day before, as well as extending to the pillars of the fauces. Again very active curettement was carried out. Smears were then made and placed in the hands of Dr. D. W. Prentiss, who made the following report:

"*Smear from tonsil;* Dr. Charles W. Richardson's case, May 21, 1904. A diplococcus resembling the D. lanceolatus more abundant than any other organism. Staphylococci fairly abundant. No bacilli having the characteristics of the fusiform bacillus of Vincent present. No spirillæ or tubercle bacilli present."

I saw the patient again on May 20th. The necrosis was not only more extensive in depth, but also in area. Some attempt was made to curette away the necrotic tissue at this visit, but the hopelessness of the case, the weakness of the patient, and his pleas caused us to desist. The patient died from exhaustion June 1, 1904.

I shall here incorporate an abstract of the three cases previously reported in the literature:

ABSTRACT I.—The case was that of a negro man, aged forty-five years, who was admitted to the Roosevelt Hospital August 13th. Indications are given in the history, which is quite full, of a sore throat first noticed on the 18th. The patient was rebellious against the use of the depressor, and little was seen in the pharynx. Note of August 20th recites the death of the patient from hemorrhage from mouth and nose. At the autopsy "a large ulcerated area was found in the pharynx occupying the site of the right tonsil and extending for some distance on the postpharyngeal wall; to this a large slough was attached by one extremity. The odor was very offensive."—"Gangrenous Tonsillitis, Fatal Hemorrhage," by E. B. Cragen, *New York Medical Journal*, September 1, 1888, vol. xlviii. p. 233.

ABSTRACT II.—The patient was a young man, aged twenty-six years. Had been attended a week for a sore throat when seen by Dr. Howe. "He had the peculiar pallor and partial stupor or lethargy of considerable toxæmia. The stench in the room was

simply fearful, and at once suggested some necrotic process." At the beginning he had the ordinary symptoms of a peritonsillar abscess. There was a fetid, brownish fluid issuing from his mouth. Deglutition was almost impossible from the great pain; temperature, 104.4°; pulse from 110 to 120. "On looking in his mouth, I found his gums, the inner surface of the labia, and the under surface of his tongue greatly swollen and covered with deep excoriations or ulcers. Both tonsillar organs were swollen and of a deep red or purplish color, indicating considerable stasis. On the left side was a large tumor that involved the entire tonsillar region, extending beyond the median line nearly to the opposite tonsil and well up into the nasopharynx. On the anterior surface of this tumor was a circular necrosed spot about half an inch in diameter, of a peculiar greenish color of gangrenous tissue, and in its centre was a perforation through which came a dark brownish, grumous discharge." The mass, when removed, was black and firm, and could be sliced much as putty or moulding clay can. It had all the appearance of having undergone a dry, gangrenous process. The odor was intense. "The removal of the tonsil exposed a large cavity lined with half an inch or more of greenish-black, necrosed tissue." The treatment was cutting away gangrenous tissue, spraying with hydrogen dioxide and the application of nitric acid. By this procedure the gangrenous tissue was entirely removed and patient recovered in about three weeks. About three months later the patient succumbed to another attack.—Report of a "Case of Gangrene of the Tonsil," by Alexander C. Howe, *Philadelphia Medical Journal*, March 17, 1900, No. 11, vol. v. p. 613.

Abstract III.—The case described by Dr. McVeigh occurred in a girl, aged ten years. The gangrene affected both tonsils. The odor emitted was the characteristic gangrenous stench, which, from his description, must have been very penetrating. The diseased condition had existed for several weeks before coming under his care. The child succumbed one week after being under his observation. He discusses the case, and, from his remarks, we can readily judge that he was alive to the nature of the case and its differentiation from the tonsillar ulceration of scarlatina, diphtheria, measles, and abscess.—"Gangrenous Eschar of the Tonsils, Death," by John Francis McVeigh, *Dublin Quarterly Journal of Medical Science*, 1867, vol. xliv. p. 78.

Remarks. When preparing the report of my first case of gangrene of the tonsil, I had occasion to look up the literature on the subject and was quite surprised at the small number of reported cases. While preparing this paper I again carefully searched the cards of the Surgeon-General's library on the subject of the tonsil, with the result of finding only the three cases which I have above abstracted.

In my first case, on account of the circumstances attendant upon the location of the patient and his rapid death, no satisfactory study

could be made as regarded the exact method of infection. Every effort was made to trace out the source of infection in the second case—the result was as fruitless as that in the first case. The patient was in perfect health at time of infection, giving daily attention to his business. All organs of the body were functionating normal. Neither in his domicile nor in the food ingested could the source of infection be traced. The bacteriological examination does not furnish us with much positive information as to the infecting organism. A diplococcus is not the usual type of organism producing gangrene, yet it is the one most abundantly found in this case. The investigations are of more value in a negative way—we know that there were no fusiform bacilli of Vincent's, no Klebs-Loeffler or tuberculous bacilli. I was greatly disappointed with the laboratory investigation. The most impressive feature of this second case was the intense and penetrating odor of the necrotic tissue. It is also interesting to note in the two cases reported by me that the one represents the moist and the other the dry type of gangrene.

Unfortunately, no autopsy was attainable in either of the two cases which were under my observation. In the first case, for various personal reasons, it was not suggested; in the second I was, unfortunately, absent from the city at the time of the patient's death.

THE PREVENTIVE AND REMEDIAL TREATMENT OF ACUTE RHEUMATIC ENDOCARDITIS.[1]

By Beverley Robinson, M.D.,
of New York.

No practitioner of medicine who has had a dispensary or hospital service can help having an earnest purpose and wish to prevent, as far as possible, the development of acute rheumatic endocarditis. These feelings arise because of the keen appreciation, not of the great seriousness at the time of many cases of this disease, but of the woful and usually irremedial results which follow it. The larger number, as we well know, of all chronic cardiac disorders proceed directly therefrom, and when valvular disease is once established our power to cure organic lesions which are present is lacking, and even our ability to restrict the ultimate outcome of stenosis, insufficiency, or myocardial changes is often much limited. It is evident, therefore, that the study of prophylaxis in this affection is most important and far-reaching. We should effect prevention, not by directly combating the onset of endocarditis, but rather by

[1] Read by title before the Climatological Association, June 30, 1905.

aborting the attack of rheumatism which so often causes it. To-day, acquired knowledge points in many instances to the throat as being the door of entrance of the rheumatic poison. This statement is especially true of children. In diseased tonsils have been found streptococcus which, inoculated in animals, has produced articular rheumatism.[1] Further, Dr. F. A. Packard[2] reported five cases of endocarditis following acute angina. In three of these cases it was definitely known that the heart was normal before the attack of angina. During the attack endocardial murmurs developed; after the attack cardiac signs and symptoms persisted. This same author cites numerous instances reported by other observers, where cardiac lesions followed acute amygdalitis. Mention is also made of the frequency of cardiac lesions without the history of preceding rheumatism, but with the history of one or more attacks of severe sore throat. Sometimes the tonsillitis is present at the same time with other rheumatic manifestations, and it is difficult or impossible to affirm that the throat was the portal through which the rheumatic poison entered the system. Such a case, it seems to me, is one referred to by Dr. N. E. Brill,[3] where numerous cutaneous manifestations, together with involvement of several joints, followed the tonsillar inflammation. In this case a "blood culture was made and diplococcus tenuis was found, which, when guinea-pigs were inoculated, gave rise to arthritic pains and injured the valves."

To keep the throat in a very healthy condition is, therefore, a primary and essential indication. Not only is acute endocarditis thus prevented: if chronic endocarditis exists as a result of rheumatic infection, we can prevent in this way a renewal of endocardial inflammation. And even if sometimes we fail to prevent the development of tonsillar disease and the ensuing rheumatic attack, at least we may help the heart in a measure to withstand the invasion, and this in a sense is prophylaxis, according to Babcock. Tonsillar inflammation should be carefully and efficiently treated by local applications, and not only should acute manifestations be thus properly cared for, but all recurrent and chronic forms of nasopharyngeal, faucial and buccal disorder should be so governed that soon a normal condition of organ may be attained.

Whenever the use of sprays, gargles, and local pigments are unavailing, appropriate use of mild cauterizing agents is absolutely essential. Indeed, in some instances of enlarged, indurated tonsils, where the crypts are filled with foul-smelling, cheesy deposits, the microbic disease is not fully eradicated, short of operative procedure, followed by appropriate topical medication.

But general conditions must not be, and, indeed, cannot be,

[1] Article by Babcock in Medicine, March, 1901, p. 209.

[2] New York Medical Journal, April 29, 1905, p. 852; also Meeting of Association of American Physicians, May, 1899.

[3] New York Medical Record, November 12, 1904, p. 795.

ignored if we may expect the best results. The gastrointestinal tract is to be kept in an aseptic and fluent condition. All fermentations proceeding from careless, injudicious eating or dietary should be arrested with correctives or well-selected antiseptic agents. Satterthwaite[1] points out the correlation between intestinal disturbances and endocarditis, and speaks of instances where "in the course of an obstinate diarrhœa there was a sharp attack of endocarditis," and adds that "in the light of our present knowledge we may attribute it to gastrointestinal disorders."

But now comes an even more important condition to be considered and treated. This is the present and underlying rheumatic toxæmia. To prevent an outbreak, attention to diet, clothing, habits, surroundings, and occupation are very important. Good nutrition should be kept up by sufficient and suitable food. Wet and cold should be avoided. A suitable antirheumatic drug, or combination of drugs, should be taken immediately. And our choice seems at present to be confined to one of the salicyl groups, with or without alkalies or alkaline diuretics. Of the former, salicylic acid, one of its salts, and preferably the salicylate of soda, or ammonia, oil of wintergreen, or the synthetic methyl salicylate or salicin, the alkaloid from the willow bark, comprise the principal drugs which have received widespread commendation. Bicarbonate of potash or soda are the alkalies most employed; citrate, acetate, and nitrate of potash are the alkaline diuretics and refrigerants.

If the tonsillar inflammation is moderate and acute, small doses, three or four times in twenty-four hours, of some one of the antirheumatic remedies, is all that is required, and soon the beneficial results from their internal use is very obvious. The acute inflammation quickly subsides, and locally the mucous membrane again seems normal. If, however, the acute inflammation is simply grafted on a preceding condition in which chronic structural lesions are present, we shall in a few weeks or months have a recurrence of the disease, and it is only after more radical measures have been instituted that a permanent cure is obtained. In some of these instances the digestive organs already much disordered for a long while require particular attention in the manner already indicated. Whenever evidence of endocarditis, usually wholly objective and reduced simply to the expression of a systolic bruit at the apex, has been discovered and apparently developed without adequate cause, a neglected throat trouble has at times appeared to me clearly explanatory.

The first threatening outbreak of rheumatism may be that of the acute febrile disorder. So soon as the nature of the disease is defined antirheumatic medication should be immediately begun. The treatment at this time is most important. Not only should the right

[1] Diseases of the Heart and Aorta, New York, 1905, p. 42

drug be selected, but it should be given at proper intervals and in sufficient dose. I have come to the ultimate conviction, partly by personal experience, partly by observation and analysis of my colleagues' hospital treatment, and in view, also, of my appreciation of what I have read, that no antirheumatic remedy is so useful and so free from objections as salicin. While I *am not prepared by my* own observation in acute rheumatism to endorse unqualifiedly the more than emphatic encomiums of Maclagan, I am inclined to believe from what I do know and have seen that he is substantially correct—viz., that salicin more nearly approaches a true antirheumatic specific, without great drawbacks, than any one we possess at present. Given in anything like a similar dose, salicylate of soda or oil of wintergreen will very soon occasion unpleasant or injurious effects. Oil of wintergreen causes head symptoms, in large or frequently repeated doses, which are annoying and frequently alarming. Salicylate of soda causes stomachal intolerance and, unquestionably, cardiac weakness, more or less enduring. Indeed, in cases in which it was probable that previous myocardial changes were present, the evil effects of its use were undoubted and lasting. Greenhow[1] states, for example, that "marked depression of the pulse and action of the heart" followed its use in almost every case—fifty in all. Further, he states it does not prevent or diminish "the frequency of complications in rheumatic fever." Against salicin no such objections can be urged, and even Greenhow, who, it seems to me, is somewhat too rigid in the proper estimate of its value, has not supported them. Speaking of salicin, Maclagan[2] writes: "It produces none of the deleterious effects of the salicylates. In several cases, some of which I have recorded, I have given full doses of salicin to patients suffering from the depressing and disturbing action of salicylate of soda; and under its use (though, of course, not in consequence of it) the depressing effects of the salicylate have disappeared." Salicin has been given in doses of 30 grains every hour for eight doses, and subsequently of 20 grains every two hours for one or several days, without occasioning appreciable accidents of any kind, and apparently with only relief to the patient's symptoms and general condition.

In the cases studied by Greenhow he gave the salicylate less frequently and in smaller doses, usually, than he did salicin. On the other hand, Preble[3] affirms that the salicylates "should be pushed to intoxication in the shortest possible time," and, further, that "the existence of a cardiac lesion is not a contradiction to their use." My personal observation would not support either of these statements. I would add, however, that David B. Lees insists also upon the great importance of giving large doses of the

[1] Clinical Society Transactions, 1886, vol. xiii. p. 244.
[2] Proceedings of the Medical Society of London, 1881, vol. vi. p. 112.
[3] Progressive Medicine, March, 1905, pp. 175–176.

salicylates in the rheumatism, acute and subacute, of adults and of children, but safeguards them with bicarbonate of soda. It is clear to my mind, however, that practitioners have usually erred in not giving larger and more frequent doses of salicin, especially in the initial stage of acute articular rheumatism, and so long as fever and pain in the joints were present. Of course, as Fagge[1] says, in speaking of the salicylates, the doses and frequency of giving which is essential to achieve good results, varies much with the case and the individual, and "it is generally necessary to use a larger quantity of salicin."

By the use of salicin the poison of rheumatism may be neutralized, the disease shortened, and thus there is less likelihood to have endocarditis appear. In those cases in which we do have endocarditis after treatment has begun, it may be that salicin was not made use of soon enough, and in sufficient doses. As treatment with it does no harm, it is wise in any event to persist in giving it, and, possibly, thus prevent further cardiac complications. It has been questioned, where salicin was given and no endocarditis developed, whether endocarditis would have occurred if no salicin had been given. There are no statistics to afford convincing proof, perhaps, and the favorable cases occasionally observed may have been accidental. And, after all, outbreaks of rheumatism vary at different times. It may be added, however, that "one[2] may reasonably expect that any remedy which has the power of arresting acute rheumatism, so that after its administration fresh joints no longer become affected, must also hinder the development of what is believed to be an analogous morbid process in or around the heart."

I believe salicin is such a remedy. Indeed, the report which I have from Dr. J. E. Traub, my former house physician at St. Luke's Hospital, New York, is to the effect that salicin has there proved most satisfactory of the salicyl group in the treatment of rheumatism, but as to its effect in preventing or curing acute rheumatic endocarditis, there was no evidence of a convincing kind to be obtained. Kinnicutt[3] believes that his cases treated with oleum gaultheria presented less than the usual proportion of heart complications. The late Dr. Flint,[4] Gottheil,[5] and Seelye[6] have reported analogous results. Greenhow[7] writes that salicylate of soda does not prevent or diminish "the frequency of complications in rheumatic fever."[8]

[1] Practice of Medicine, p 557

[2] Fagge Practice of Medicine Philadelphia, 1886, vol ii p 537

[3] Medical Record, vol xxii p 505

[4] Philadelphia Medical Times, vol xiii p 846.

[5] Medical Record, vol xxiv p 258

[6] New York Medical Journal, November, 1884; National Dispensatory, 1894, p 1125

[7] Clinical Society Transactions, 1880, vol xiii p 345

[8] Methyl salicylate, or synthetic oil of wintergreen now used in one New York Hospital by reason of false economy, is I believe, an undesirable substitute for the natural oil In many cases it is tolerated even less well by the stomach than salicylate of soda. According to latest information it may be that pure ethyl salicylate possesses several advantages over the natural as well as the artificial methyl salicylates It is said to be less toxic than oil of wintergreen, and less irritating to the stomach than salicylate of soda (June 23, 1905)

In cases of recent valvulitis, Richard Caton[1] advocates strongly the use of iodides, "partly to help in the absorption of morbid exudations, but chiefly to lower muscular tension." He endeavors also to influence the cardiac vasomotor and trophic nerves reflexly, by gentle and almost painless stimulation of the first four dorsal nerves which we know to be in close relation with the heart. Babcock[2] is unwilling to accept any other evidence of the cure of acute endocarditis than what proceeds from autopsy findings, and his experience is not sufficient hitherto to enable him to be convinced of its possibility. "The mere disappearance of a cardiac murmur is not sufficient to justify a conviction in this regard." Clinical observation and pathology show, writes Babcock, that once acute endocarditis is thoroughly developed we have no means to cure it.

In instances of subacute and chronic rheumatism, where I have given salicin many times in small or moderate doses, more or less frequently, I have repeatedly known symptoms to disappear completely, or be much ameliorated in a few days or weeks, and without any intercurrent manifestations of an unfavorable nature which could be attributed to its use. I cannot say as much of the salicylate of soda, but shall make an exception for the ammonia salt which has stood me in good stead oftentimes, and particularly whenever there was marked stomachal irritability. We must ever bear in mind that while the heart may be attacked at the same time as the joints, we are not aware of it simply because it takes several days for the inflammation to be propagated from the fibrous to the serous layer, and then only through thickening and friction of the valves, occasion the condition which gives rise to an audible murmur—viz., minute or warty vegetations. The importance of this fact should be insisted upon both in the acute and subacute forms of rheumatism. Even when fully developed, Maclagan believes in the cure by means of salicin treatment and prolonged absolute rest of many cases of acute endocarditis. The importance of this rest cannot be insisted upon too strongly during the entire attack of rheumatic fever. Rest must be both mental and bodily. Thus the heart is freed additionally from sources of much increased action and tension, and it is evident in this way we lessen somewhat the chances of cardiac derangement.

In many of the ailments of children and adolescents, where they are closely watched, a marked rheumatic dyscrasia will be, if not at once, finally discovered. These affections differ materially with those of adult age. Articular symptoms are very infrequent; cardiac complications are the rule. Indeed, sometimes an endocardial murmur with fever is almost the first symptom of rheumatism

[1] Lancet, February 4, 1905, p. 301.
[2] Diseases of Heart and Arterial System, 1903, New York, Appleton & Co.

which is manifest. With it, however, may appear cutaneous symptoms, such as those which characterize erythema, purpura, recurring urticaria, or subcutaneous nodules. These nodules are occasionally seen over a superficial bone or tendon. Accompanying them there may be no fever and no articular manifestations. For Cheadle, the cutaneous disorder, accompanied by the endocardial bruit, is always a condition involving a very grave prognosis. If the rheumatic origin of any of these signs be recognized, we should save the heart from unnecessary strain and prevent, if possible, its becoming involved. Sometimes, despite rational precautions of prophylaxis, there may be an outbreak of acute articular rheumatism, and the heart may become affected. Indeed, this is so frequent as to seem to some observers a part of the normal march of the disease with children, and not a complication of it. It is most important to recognize it as soon as it does exist. This we may often do by a murmur or muffling of the first sound of the heart at the apex. There may also be an accentuation of the second pulmonic sound, or a reduplication of the second sound at the apex. (Parkinson.[1]) Irregularity and inequality of the pulse are deceptive, as they may occur in health. If our diagnosis of the presence of rheumatism be made, it should be promptly treated; first, by a proper amount of one of the salicyl group, and best of all by salicin; second, by absolute or relative rest of body and mind, depending upon the degree, stage, and locality of the rheumatic affection.

In giving the specific treatment of rheumatism, with salicin or a salicylate, care should be observed not to interrupt treatment too soon, and not to reduce the amount of the drug too rapidly. In either case, if we do, a relapse more or less serious of all previous symptoms is liable to occur and to last a longer or shorter time. What is true of one relapse is true in a very measured degree, unfortunately, of two or even more relapses. I am satisfied that with children especially, and when the heart becomes involved, as is usually the case, the salicylates cannot be persisted with in even moderate doses for a continued period, without doing considerable irremediable harm. In these instances I would insist most emphatically upon the proper use of salicin as being a far preferable, because a safer, remedy to administer. It may not act always so rapidly and at first, apparently, so usefully, but if our observations be careful and continued I am convinced all will agree with me and share my conviction. Despite treatment with salicin, however, these cases go frequently from bad to worse, and many of them result fatally, owing to an advanced condition of heart disease. While this statement is unfortunately true, I am confident more such cases are hurt unduly where salicylate of soda has been given to combat pain and lessen fever, by reason of its depressing action

[1] The Hospital, October 3, 1903, p. 10.

upon a heart with a myocardium already relaxed, softened, weak, and showing signs of beginning degeneration. In such cases it is wisdom frequently to diminish the amount of the salicylate and even of salicin, and combine with it a suitable amount of alkali. Dr. Gilbert Smith's tables, indeed, seem to show that the percentage of heart complication, at St. George's Hospital, London, "might be due to the fact that the practice of combining full doses of alkali with the salicylate was general" at that hospital. Indeed, the percentage was lower than at the other hospitals, and also than the general average of the presalicylate cases. This is in line somewhat with the statements of Fuller and Dickinson, who had shown that alkalies had a great influence in preventing heart complications.[1] The old alkaline treatment was essentially eliminant, and in my judgment, even if it does not shorten rheumatic attacks in adults or children, or prevent or cure endocarditis, usually, at least, it does no evident harm. Pursued moderately, during several days or weeks, and especially when we give therewith a suitable cardiac tonic or stimulant, such as sparteine, caffeine, or strophanthus, it relieves symptoms frequently, and without injurious after-results. David B. Lees[2] urgently advocates the necessity of combining with the salicylates at least double the quantity of bicarbonate of soda in order to prevent salicylate acid poisoning and the "air hunger" so characteristic of it.

For my own part, feeling as I do about the utility of biliary disinfection in the successful treatment of rheumatism, and recognizing the probable power in this direction of the salicylate group, and notably of salicylic acid, I am inclined at times to combine the use of chloride of ammonium with an alkaline diuretic like acetate of potash. I have assuredly seen more than once fever and pain lessened very notably by this combination, and in cases, perhaps, where the salicylates had been used already, without good results. The chloride of ammonium has been recognized for many a year as an hepatic stimulant and antineuralgic, among the most valuable. The late Dr. Anstie, of London, has interested me much, through his work on neuralgia, in the use of this salt. And a fact worthy of note is that the combination to which I refer was a favorite one with a down-town practitioner of New York, who, twenty to twenty-five years ago, had a very extensive reputation, even among the wealthy and cultured classes, for his great success in treatment of rheumatism in its subacute and chronic forms, of men and women, of middle life or beyond.

In acute exacerbations of the disease tincture of aconite was added with very great advantage to the relief of symptoms and comfort of the patient. The aconite was suppressed as the patient improved. In another toxæmia, namely, that of influenza, the

[1] Medical Society Transactions, vol. vi. p. 125.

[2] Acute Visceral Inflammations, London, 1901, p. 296.

alkaline treatment with chloride of ammonium is sometimes useful also, and particularly if there is any painful expression of the disease in connection with an endocardial murmur.

When any of the abnormal signs of rheumatism appear in a child, he or she should be confined to bed. And even though the child seems very little ailing, he must be kept there until all signs of rheumatic trouble have passed, either of heart or other organ. It is a small matter, writes Ashby,[1] to be overcareful in keeping in bed a child with slight joint trouble, but it is a grave affair to ignore acute endocardial trouble and to let children run about too soon. Usually endocarditis in children is accompanied also with myocarditis, and this is an additional reason for insisting upon rest in bed—viz., physiological rest. Indeed, there may be both pericarditis[2] and myocarditis, and thus all the heart structures are soon involved. Rest is even more important with children than with adults, as we know with them how liable the heart is to be attacked by the rheumatic poison, and how serious the remote results of it are. The task of absolute and prolonged rest in bed for children is a very difficult and ungracious one many times, but may occasionally be accomplished completely and efficiently, and ultimately with happy results. If the patient is made to understand in the beginning the great importance of this rest treatment, and how only in this way a complete cure can be expected, he or she, with wise parents, will submit to it. But it requires faith, courage, intelligence, will-power, and self-control on the part of physician, parent, and also, in a measure, of patient, to secure a thoroughly satisfactory result; I mean a complete cure. To no one more than to the late Dr. Maclagan, of London, and, latterly, to Dr. Caton, of Edinburgh, are we indebted for their wise insistence upon the all-important necessity of absolute rest in the treatment of acute endocarditis, at a time when it is yet possible to achieve a complete and permanent recovery. The guiding hand of the physician to prevent overstrain of the heart must be used not only during the period of absolute rest, but continued a long while afterward. This continued care and watchfulness is far more important in reality than the treatment of cardiac inadequacy due to permanent lesions when it has once developed. We perceive, then, that the physician's duty is not terminated when acute cardiac inflammation has disappeared. He must prevent chronic cardiac disease of valves and muscle and incompetency resulting therefrom, by wise counsel and avoidance for the patient of overfatigue. There is much for a careful physician to do in this direction, and he fails in duty if he does not instruct his patients who have had acute endocarditis what

[1] Ashby and Wright. Diseases of Children.

[2] *In fatal cases, out of 150 only 9 escaped pericarditis, and at least one-fifth of the number* had myocarditis. (Poynton.)

to do and what to avoid. Here, as Babcock writes, is "prophylaxis in the truest sense."

It is particularly important for patients affected with subacute rheumatism to take a complete rest for a while. In this way they will, I believe, often prevent the development of an attack of acute endocarditis. If a chronic valvular trouble already is present, this counsel of rest should be especially heeded, because if acute endocarditis becomes manifest it will surely aggravate the previous cardiac condition. It is wise to avoid all violent exercise during the period of convalescence. It is also prudent for the physician to see the patient frequently, so that the first distinct evidences of the presence of acute endocarditis may be immediately and effectively combated. Mental strain should be avoided as much as bodily effort. The former, like the latter, is apt to occasion cardiac inadequacy. This it does through augmented cardiac action caused by excitement, brain-fag, mental weariness. The slower the heart acts the longer the diastole, and hence the less violent its contractions. Unfortunately, the heart cannot be put at complete rest as the joints can, and for this reason it is very often difficult, or well-nigh impossible, to secure a complete cure. The heart should be quieted by the use of bromides internally. Thus the risk of rupture of the cusps through overaction or the transport of emboli to different organs is notably lessened. Digitalis should not be used to quiet the heart. While this drug slows heart and pulse, it increases the strength of cardiac systole and may occasion injury to the softened valves by the additional strain thus occasioned.

I confess I am somewhat skeptical in regard to the good effects of any local application in preventing endocarditis, unless combined with intelligent general treatment. In any event, as a preventive measure, I would not apply leeches, blisters, or even an ice-bag, with this object in view, unless there were rather pronounced symptoms indicating their use. The slight advantages possibly connected with their employment would be more than balanced by the annoyance which they would occasion. As regards the remedial effect of local applications, when acute endocarditis already exists, I hold somewhat different views. Whenever there is weakness and irregularity of pulse and heart, it is desirable to employ hot fomentations, and these, I am confident, are far better than cold compresses or an ice-bag, as their action is decidedly more stimulating. I am also satisfied that repeated fly-blisters over the precordial area are distinctly useful in a remedial way in commencing valvulitis, and, despite any nervous distress or pain occasioned by their employment, would insist upon them. Iodine, mustard, turpentine stupes, etc., do not take their place or act so beneficially.

In this connection I would cite James Tyson,[1] who believes "if

[1] Transactions of the Association of American Physicians, 1897, vol. xii, p. 203.

the blister is applied sufficiently soon, we may even avert the effusion" in pericarditis. Likewise, in endocarditis I believe we may avert roughening of valves and fibrinous deposit on them. Lees objects to the use of fly-blisters, on the ground that the soreness produced by them interferes with the physical examination of the heart. He admits, however, that they may be moderately useful in causing local muscular depletion. From this point of view he regards them as far inferior in value to the use of leeches. These, followed by the application, more or less continuously, of the ice-bag, diminish blood pressure in the right auricle, and when this portion of the heart is dilated, lessen its size and relieve symptoms very much, particularly dyspnœa and some lividity. An occasional mercurial is, in my judgment, advantageous. I do not believe aconite, veratrum viride, or tartar emetic are often indicated. In favor of the use of aconite I would say, however, that when the acute endocarditis is grafted on an old valvular lesion with general arterial changes and marked hypertension, it may exercise a valuable restraining influence in quieting the heart. It must be managed even then, however, with great care and good judgment, and not carried to the point where notable general depression followed its use.[1] What remains of treatment pertains mainly to allaying symptoms as they arise, and without having in view any evident and direct remedial effect. Thus pain and restlessness are combated with opium, or in children, preferably, with the bromides. Soothing liniments, locally, may also be advantageous. I rarely, if ever, make use of the coal-tar antipyretics, and, except phenacetin in very small doses, believe they invariably do harm. High temperature seldom exists or calls for hydrotherapy. The latter is indicated, as a rule, merely for the patient's cleanliness and comfort.

In the use of strychnine, where there is evidence of myocardial degeneration, I now rarely give doses beyond $\frac{1}{60}$ grain four times in twenty-four hours, as I believe, with Dr. Curtin and Dr. Jacobi,[2] the good effects are questionable with large amounts. Opium, in many such cases, when given in moderate doses, is a far preferable supporter of the heart. (Musser.[3])

In some of these cases of systolic bruit at the apex during acute or subacute, or even chronic rheumatism, we are in doubt for a time, shorter or longer, as the case may be, as to whether the cardiac murmur really proceeds from or is caused by the diseased endocardium, or is hæmic, or due to a dilated orifice caused, perhaps, by anæmia, by nervous derangement, or relaxed and softened valves, or cardiac muscle. Time, judicious treatment, prolonged rest, absolute or relative, will solve the diagnostic diffi-

[1] During the past winter I saw a case, in consultation, where aconite was undoubtedly useful, and particularly in affording relief to distressing cerebral symptoms.

[2] Discussion of Dr. Musser's paper.

[3] Paper read before the Association of American Physicians, Washington, May 16, 1905.

culties many times, and in any event are usually the best remedial means for a while, which we can rationally advise and, indeed, strenuously urge. With returning health both murmur and dilatation may disappear because the heart has regained its tone. Autopsies have shown that "one out of seven cases in which heart murmurs are heard no endocarditis is present. In about one in seven cases in which endocarditis is found at autopsy no murmur was heard during life."[1] Again, as Jacobi[2] wisely states, "often we were apt to make a diagnosis of endocarditis when there was a myocarditis present." He had such cases under observation year after year; now and then they turned up with a systolic murmur (mitral). Then they would get better and remain so for months after treatment by rest, etc. The murmur would be brought out by the slightest exertion.

There is one remarkable and suggestive analogy in acute articular rheumatism, which has been insisted upon by more than one observer, although ignored, apparently, by many. It is the resemblance, in many particulars, to malaria. Even as regards the endocarditis we have many features which are striking and common, and in relapses of each disease the need of long continued, although at times intermittent, medication to achieve the best curative results.[3] Singular to say, the willow tree is most prolific in the soil and under ambient conditions where rheumatic toxemia would most probably be furthered by secondary causes, just as the tree from which cinchona and its alkaloids are obtained is found in countries where malaria is endemic in most pernicious form, and just as coca comes from a region where the natives particularly require the staying power to ascend great mountain heights which mastication of the fresh leaves of the plant surely gives.

In rheumatism, in influenza, in cerebro-spinal fever, and still other infections, similar symptoms often proceed, no doubt, from different microbes. It is possibly this latter element of the disease that explains, in some sense, at least, the alleged curative property of the diphtheria antitoxin in the late outbreak of acute meningitis.

"In view of the favorable results in some of the cases of mixed infection, one or more of the accidental toxins may have been neutralized, while the principal one that happened to be inconsiderable in virulency and possibly untouched was more easily overcome by a natural vital resistance of the patient. Such an assumption would give a reasonable explanation for the cases that proved fatal in spite of the new treatment."[4] It seems to me that this thought applies equally well to what we know to-day about

[1] Medical News, May 20, 1905, p. 958. [2] Medical Record, May 20, 1905, p. 1905.

[3] In an able paper, Dr. Warren Coleman has lately pointed out close resemblance also between malaria and infective endocarditis. (THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, March, 1905.) Query to my mind and to others is whether all cases of endocarditis are not infective. See Brill's case.

[4] New York Herald, April 7, 1905 (editorial).

the bacteriology of rheumatism and its frequent, at times almost essential, complication—acute endocarditis. This pathological condition occurs in a great many diseases, and it is a well-established fact that it follows many bacterial infections—syphilitic, pneumonic, streptococcic—after scarlet fever, rheumatism, and many others.[1]

Hence, I am of the opinion, and my clinical experience would seemingly corroborate it, that while salicin and the other members of the salicyl group relieve rheumatic symptoms in a remarkable manner in many instances, in other apparently similar cases they fail to do so. Why, we do not know as yet positively. In just such cases I feel confident that the rheumatic poison is still present and active; but we are unable to neutralize it, or in any event, with our ordinarily most effective remedy. In view, however, of clinical facts which show the similar pathological effects of different micro-organisms, we now endeavor to give an explanation which is rational and, provisionally at least, fairly satisfactory. Take one notable example to prove it. It is well known, and has been so for many years, that a Spa treatment at sulphur springs (Aix-les-Bains, Richfield, Sharon, etc.) has been judged to have positive therapeutic value in many cases of rheumatism, in which other remedies had ceased to be of value, or even, originally, had not responded favorably to our legitimate anticipations. Aside the theory of the causation of rheumatism, based essentially on the fact that it is due to the action of "diplococcus tenuis" or other form of micro-organism, it is evident that provisionally "we must be content to combat the symptoms of the disease, as they present, by purely empiric methods." (Conklin.)

In conclusion, I would emphasize again the importance of my subject for discussion—than which, indeed, I know of none of greater interest. It seems to me that to the late Dr. Maclagan and to Dr. Lees, both of London, the profession is under deep and lasting obligation for demonstration of facts fruitful of enduring advantage to innumerable patients.

NOTE.—During the past winter and spring I have suggested the use of salicin in the treatment of epidemic cerebrospinal meningitis. In several cases, at different hospitals of New York (St. Luke's, Bellevue, Gouverneur, etc.), salicin was given a trial, and, while it did not yield notable remedial results as regards the outcome of the disease, it did show marked power over the joint symptoms manifested in connection with it in a few instances. I am glad to note that apparently good results have been obtained latterly by Dr. A. Seibert[2] in cerebrospinal meningitis from rectal injections of large doses of sodium salicylate.

[1] Brill. New York Medical Record, November 12, 1904, p. 795.
[2] Medical Record, June 17, 1905. p. 930.

CHYLOUS AND CHYLIFORM EFFUSIONS INTO THE SEROUS SAC: WITH A CASE.[1]

By J. G. Wilson, M.D.,
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The subject of effusion of chyle, chyle-like and fatty fluids, has been especially investigated in this country by Busey, whose papers contain full studies of the early literature of the subject and important collections of cases. The paper read by Dr. Busey before this Society in 1880[2] contains a table of 33 collected cases of "effusion and accumulation of chyle and chyle-like, milky, fatty, and oily fluids in the peritoneal cavity," together with abstracts of 13 cases collated after the table was prepared. In Germany, Quincke (1875),[3] Bargebuhr (1895),[4] and again (1895),[5] Rotmann (1897),[6] Muttermilch (1902),[7] and others, have published valuable papers upon the subject.

To Quincke is due the credit of having first pointed out the distinction between chylous and chyliform effusions—hydrops chylosus and hydrops adiposus—while Bargebuhr recognized the important fact that a nice distinction between these two forms of effusion cannot in all cases be made, and that in some cases the presence of the chylous transudate and the chyliform exudate at the same time is by no means improbable: *gar nicht undenkbar sei.*

Bargebuhr collected 126 cases, including Busey's cases, reported between 1633 and 1894.

Rotmann's collection of 29 cases includes some that were overlooked by Bargebuhr and those published subsequently, making 155 in all to 1897. Of the latter 3 were personal observations made in Mosler's clinic. Since the date of Rotmann's paper many cases have been reported.

It is certain that the frequency of chylous and chyliform effusions is far greater than the literature indicates. The appearance of the fluid is striking, and at once arouses the attention of the trained observer; yet it presents in some cases a superficial resemblance to pus, which may mislead the physician whose attention has not been especially called to the subject. There are no means by which the nature of the effusion may be determined *intra vitam*

[1] Read by title at the Twentieth Annual Meeting of the Association of American Physicians, Washington, D. C., May 16, 1905.

[2] Transactions of the Association of American Physicians, vol. iv, p. 76; The American Journal of the Medical Sciences, vol. xc, p. 373.

[3] Deutsches Archiv f. klin. Med., Bd. xvi, pp. 121–139.

[4] Ibid., Bd. li, pp. 161–192.

[5] Ibid., Bd. liv, pp. 410–441.

[6] Zeitschrift f. klin. Med., Bd. xxxi, pp. 416–441.

[7] Ibid., Bd. xlvi, pp. 123–134.

except by the withdrawal of a portion of it. In many of the reported cases its presence was first recognized at the autopsy. The fact that cases occur much more frequently than the collected statistics indicate is evident upon the consideration of the experience of those who have written upon the subject. Senator saw 8 cases, Rotmann saw 5 cases at Greifswald in the course of two years, and I have seen in all 3 cases. Not all the cases by any means find their way into the literature, for the reason that many observers hesitate to report single cases and the cases which recover and those in which autopsy cannot be made are necessarily incomplete.

Rotmann's study of 155 cases shows the effusion to have been present in the abdomen in 104 cases, in the pleural cavity in 49 cases, and in the pericardium in 2 cases. In certain cases, as in that which forms the basis of this communication, the milky effusion has been found both above and below the diaphragm.

The pathological causes of milk-like effusion into the serous sacs are, in general: (1) trauma; (2) tuberculosis; (3) malignant disease; (4) filariasis; (5) thrombosis of the left subclavian vein; (6) disease of the liver; (7) syphilis.

The particular causes of such effusions into the peritoneal sac (*chylous ascites*) comprise those conditions which produce a solution of continuity in the vessel walls by injury, inflammation or extreme tension, or by transudation through the wall weakened by disease. Under this heading Rotmann has grouped the following special causes in the order of their frequency: Compression of the thoracic duct or the lymphatic vessels by glandular enlargements, neoplasms, etc., 17 cases; non-tuberculous peritonitis, 9; occlusion of the left subclavian vein, 6; excessive pressure, strain, cough, 5; peritoneal carcinoma, 5; filariasis, 3; occlusion of the thoracic duct, 3; occlusion in lymph glands, 3; occlusion of lymph vessels, 2; external pressure, 2; malignant lymphoma, 2; disease of the liver, 2; syphilis, 2; primary disease of the lymph vessels, angioma, 1; and calculus in the receptaculum chyli, 1.

The special causes of *chyliform effusions* into the peritoneal sac he groups as (*a*) due to fatty changes in cells, especially endothelial, and the cellular elements of new-growths—tuberculosis, 6; carcinoma, 5; cirrhosis of the liver, 3; cardiac disease, 2; sarcoma, 2; chronic non-tuberculous peritonitis, 1; and an apparently primary fatty degeneration of the endothelium, 1—and (*b*) an abnormal fatty condition of the blood—lipæmia, 1.

The special causes of *mixed forms* are, according to this observer, carcinoma, peritoneal, mesenteric, glandular, etc., 3, and tuberculous peritonitis, 1.

The special causes of *chylous effusion* into the pleural sacs are (*a*) those leading to the escape of chyle as the result of injury or disease of the vessels, namely, external violence, 8; carcinoma of the pleura, 5; occlusion of the left subclavian vein, 4; compression

of the duct by tumor, 2; malignant lymphomatosis, 1; obstruction of the lymphatic vessels, tuberculous lymphangitis, 1; rupture of the thoracic duct, 1; filariasis, 1; and [illegible], 1; the latter by the discharge of a chylous ascitic fluid into the pleural cavity by rupture of lymph spaces, 1.

Rotmann groups the causes of chylous effusions into those as to the presence of cells in the fluid, as in carcinoma of the pleura, tuberculous exudative pleurisy, and [illegible] abnormal fat in the blood [illegible].

The two cases of fatty effusion into the pleural cavity [illegible] he attributes to rupture of chyliferous vessels and the escape of chyliform fluid from the left pleural [illegible] lymph channels. In 25 of these cases [illegible] determined.

It is obvious from a review of the [illegible] considerable proportion of the cases [illegible] as to produce both chylous and chyliform [illegible] clusion is inevitable that [illegible] many milky effusions, and that [illegible] varying proportions of true chyle [illegible].

The chemical and morphological [illegible] have been especially studied by Nencki. The [illegible] of this observer were conducted upon the [illegible] ture in the case of a child aged five months, [illegible] pleural effusion followed a fall from the mother's [illegible]

The quantity of the fluid withdrawn was [illegible] the following characteristics:

A complete resemblance to milk; opaque, [illegible] whitish color, with a faintly yellowish or creamy shade, feeble alkalinity; specific gravity, 1017. Upon microscopic examination there were seen great numbers of minute, diatomic granules in active, molecular movement. Here and there were highly refractive bodies, constituting distinct points scattered separately or in groups; a few leukocytes; larger cells containing distinct, highly refractive granules and erythrocytes in very small number, and only found upon careful search.

The chemical examination, conducted upon the hypothesis that the fluid was a fat emulsion, yielded the following results:

Shaken with ether in a test-tube the fluid became less opaque. When a few drops of potassium hydroxide were previously added it became quite transparent and almost colorless. Upon the addition of osmic acid the fluid became blackish.

Microscopically, upon the addition of a 1 per cent. solution of osmic acid to a drop of the fluid, the dust-like granules assumed a dark-gray color. The same change in color was observed in the granules in the large cells. Upon the addition of ether the

dust-like granules were dissolved. They ran together and formed at first smaller, then progressively larger, and finally quite large, highly refracting globules, which, upon treatment with osmic acid solution, became black in color.

The fluid, upon standing, deposited upon the bottom of the vessel grayish-red, flocculent masses, in which were to be seen under the microscope the above-described morphological elements.

The morphological elements were almost exclusively leukocytes and in marked preponderance lymphocytes. There were present in small numbers neutrophilic leukocytes containing fat granules, together with endothelial cells and a very few red blood corpuscles.

The chemical examination yielded the following results—100 c.c. of the fluid:

Solids	8.360
Albumin	6.135
Extractives by ether	1.481
Cholesterin	0.174
Soluble salts	0.563
Insoluble salts	0.028
Extractives by water	0.132
Extractives by alcohol	0.121

The fluid was absolutely sterile; neither microscopically nor by culture upon bouillon and agar were any micro-organisms to be recognized.

The above characters of the effusion in Mutermilch's case, considered in connection with the etiology of the condition—traumatism—and the entire absence of symptoms suggestive of pleural inflammation, justified the conclusion that the fluid consisted of chyle.

Quincke was the first to divide the fatty effusions into two groups: First, those consisting of chyle from the thoracic duct, or the lacteals—transudates—and, second, those resulting from fatty metamorphosis of the cellular elements in inflammatory effusions—exudates. It was generally thought that these two forms of milky fluid could be differentiated only by the microscope. Quincke described a case in which there was escape of chyle into the pleural cavity. The fluid was like milk, with a slightly yellow shade. Under the microscope it showed minute granules of different size, but all in the form of apparently round points, or dust particles, and a very small number of white blood corpuscles.

In contrast to this chylous effusion he described the form of fatty fluid resulting from fatty changes in the morphological elements. The fluid was bright yellow, somewhat cloudy, whey-like. Upon standing there formed upon the surface a thin, cream-like layer, showing under the microscope small globules, mostly in the form of collections of highly refractive granules, less frequently having sharp borders, and still less frequently presenting the appearance of large, indistinct nuclei. In the fluid underlying this layer were

seen white blood corpuscles and large round cells, which, in consequence of differences in the amount of fat present, form possible transition forms. In this case Quincke revealed the presence of the fat by the bright-red color of cellular elements. He designated the effusions of the first category as hydropic chylous, those of the second category as hydropic adipous.

In 1885 Senator adopted Quincke's division of the fatty effusions into hydropic chylous and hydropic adipous, placing the cases in the second category as well from the fatty metamorphosis of the endothelial and other cellular elements, and the absence of fat upon the presence of cells in which the fatty change is incomplete. It is obvious that in advanced cases, in which the transitional forms may not be found, the diagnosis of the true nature of the effusion must remain obscure. For this reason Senator attached great importance to the chemical study of these fluids, and especially to the presence of sugar-reducing, boiling-stable albumin, though contain sugar constantly in appreciable quantities, is rarely recognized, while this substance is easily found in serous transudates and exudates in moderately thin exception fluids of this nature. Grape sugar, in more than doubtful traces, was believed by this author to indicate the presence of chyle. Later investigations, especially those of Paschcles and Reichel,[1] Pichardt,[2] and Bernard,[3] have established the fact that ordinary serous transudates and exudates often contain this substance in appreciable quantities, and that its presence in chyliform exudates may also be expected.

Strauss[4] was able to demonstrate the chylous nature of the fluid in fatty ascites by giving his patient a diet rich in fat, the administration of which was found to greatly increase the proportion of the fat in the peritoneal fluid. This observer reported the rapid renewal of the fatty fluid having always the same peculiarities as indicating its chylous nature.

The milky effusions into the serous cavities do not always owe their characteristic appearance to fat. Micheli and Mattirolo[5] have described pseudochylous forms of ascites, and have satisfied themselves experimentally that in the cases which they had under observation the milky appearance arose from the presence of lecithin.

Dr. Edsall reported at the last meeting of the American Pediatric Society a case of Hodgkin's disease, not yet published, in which there was a non-fatty pleural effusion, the opacity being due to altered globulins.

The case about to be described, brought to me by Dr. Housekeeper, of Vandengrift, Pennsylvania, illustrates one of the more

[1] Charité-Annalen, 1886, p. 207.
[2] Berl. klin. Wochenschr., 1897, p. 841.
[3] Archives de physiol. norm. et pathologr., 1886, p. 207.
[4] Wien. klin. Wochenschr., 1900, No. 3, p. 56.
[5] Wien. klin. Wochenschr., 1896, No. 17.
[6] Munch. med. Wochenschr., 1896, No. 6.

common forms of fatty effusion into the serous cavities. It may, I believe, be properly regarded as an example of that group of cases in which the effused fluids, though fatty, are neither chylous nor chyliform or chyloid, but a mixture of both. This opinion is based upon the following facts:

1. The constant gross physical characters of the peritoneal fluid and that removed from the left pleural cavity upon repeated aspiration on several occasions at comparatively short intervals of time.

2. The practically identical microscopic and chemical characters of the two specimens of left pleural effusion examined by Dr. Kelly and obtained within a period of twelve days.

3. The presence in those fluids of fine dust-like granules in great numbers, and showing active molecular movement and lymphocytes, together with polynuclear neutrophiles, nuclei, and endothelial cells in various transitional stages of fatty change.

4. The different gross and microscopic characters of the fluid contained in the circumscribed right pleural effusion.

5. The underlying pathological conditions, namely: diffuse sarcoma of the retroperitoneal and thoracic lymph glands and inflammatory pleural changes.

6. The softened condition of the lymph glands in the neighborhood of the receptaculum chyli, and the fact that the walls of that structure could not be recognized upon careful search and the thinned walls of the thoracic duct, changes permitting the escape and transudation of chyle.

7. The inflammatory changes about the sarcomatous glands and the pleural inflammation, the source of proliferation of endothelial cells and serofibrinous and seropurulent exudate, with fatty changes in its cellular elements.

Sarcoma of retroperitoneal and thoracic lymph glands; fatty ascites discovered upon operation for hernia; double pleural effusion, fatty upon the left side and presenting upon repeated aspiration, at intervals extending over a period of many months, the same appearance; circumscribed and seropurulent upon the right side; microscopic and chemical notes on the fluids; autopsy.

Anamnesis.—A. M. B., female, aged thirty-five years, single, a teacher; admitted to private room in the German Hospital December 5, 1904.

Family history good; father living and well; mother died about the menopause from disease the nature of which is unknown to patient; only two brothers, who are living and in good health; no sister. No history of tuberculosis or malignant disease could be obtained.

The patient states that she had scarlet fever in childhood, but cannot tell whether or not she had the other diseases of childhood. She had a severe attack of tonsillitis at the age of fifteen, but always

regarded herself as having good health until about three years ago. Until that period she had a good appetite and digestion and regular bowels. First menstruated at fourteen, and that function has been regularly and painlessly performed until the present time. Dates her illness from the appearance of a femoral hernia upon the right side in the spring of 1902. This speedily began to give trouble, which led to an operation shortly afterward. In reply to a note of inquiry which I wrote to her surgeon, Dr. J. J. Buchanan, of Pittsburg, I received the following reply: "I operated on Miss A. M. B. for the radical cure of a femoral hernia with irreducible omental contents, if I remember aright. The peritoneum contained a considerable quantity of free fluid which was opaque, milky, or deep straw color. I remember that she was turned on her side to allow it to flow out. Perhaps there was a pint or two. No tubercles were on the sac or presenting omentum. She had no fever before or after the operation. Her wound healed *per primam.*"

The fluid was thought to be that of a tuberculous peritonitis. I was unable to ascertain whether or not a systematic examination of it had been made.

The following autumn she began to be short of breath upon exertion and had during the winter four severe attacks of bronchitis. In July, 1903, she had another attack of bronchitis. In the intervals between these attacks she had occasional slight cough and little or no expectoration, but felt constantly oppressed and was very short of breath upon even moderate exertion. At the close of the last attack of bronchitis the presence of a left pleural effusion was recognized and paracentesis was performed and a quantity of fluid, milk-like in appearance, withdrawn. This operation has been repeated a number of times, in all, as follows:

1903. July	quantity withdrawn about		1000 c.c.		
1904. July	"	"	"		1000 "
1904. October 1	"	"	"		2000 "
1904. October 15	"	"	"		2550 "
1904. November 1	"	"	"		2500 "
1904. December 2	"	"	"		2500 "

The fluid always presented the same appearance and corresponded, according to the patient, to that which was removed after she came under my observation, and is fully described in Dr. Kelly's report which follows. During this period the patient occasionally noticed a slight discharge of similar fluid from the scar in her thigh resulting from the operation for the radical cure of the hernia. Her urine has never been milky in appearance. About the time that the first aspiration of the chest was performed her cough ceased and has not recurred. She has had no chills, fever, night-sweats, headache, or pain. Two years ago she weighed 128 pounds. Her present weight is 108 pounds.

Status Præsens. A tall, slender, well-formed woman of good color, but slightly cyanotic on exertion. No dropsy or œdema; no

cutaneous eruption. Temperature normal, at times slightly subnormal; chest somewhat flattened on both sides, but more upon the left than upon the right. Respiratory movement shallow, the excursus being much more limited upon the left than upon the right side. Respiration frequency while at rest 20, upon slight exertion rapidly increased to 30 to 36.

The impulse of the heart not well made out either upon inspection or palpation. It is interesting to note in view of the postmortem findings that careful palpation and auscultation failed to reveal any signs of a mitral stenosis. In the precordial area there was no thrill, nor was any murmur to be heard. There is accentuation of the second sound at the base, particularly in the pulmonary area. The pulse is small and of moderate tension. Its frequency varies in repose from 80 to 96.

The percussion resonance is clear on both sides anteriorly in the infraclavicular regions, with a slight tympanitic quality upon the left side. Below this it is impaired upon the right side and there is dulness below, with flatness at the base upon the left side. The percussion signs posteriorly correspond to those in front except that dulness (flatness) extends much higher and there is flat tympany at the upper part of the chest. There is partial obliteration of Traube's semilunar space.

Upon auscultation feeble vesicular and bronchovesicular respiration over the upper lobes, but no rales. At the right base posteriorly a few faint subcrepitant and crepitant rales, especially upon deep inspiration. Over the area of dulness on the left side absent breath sounds diminished vocal resonance. There is absence of vocal fremitus. No friction sounds except in a limited area at inner border of left scapula. The area of liver dulness not enlarged. Its lower border corresponds to the margin of the ribs in the midclavicular line and descends about two fingers' breadth upon full inspiration.

The lower border of splenic dulness is below the costal margin.

The abdomen is soft, elastic, and symmetrical. The presence of ascites cannot be made out.

Urine amber, acid, 1030; no albumin; no sugar; phosphates, epithelial cells, leukocytes, and bacteria. Amount measured 400 to 900 c.c. Some lost at stool.

Blood: hæmoglobin, 90 per cent.; erythrocytes, 6,325,000; leukocytes, 7200.

Repeated examination both by day and by night showed no filaria.

Operation December 19, 1904, by Dr. Deaver. A large portion of the thoracic fluid was removed with aspirator. An incision 12 cm. long was made parallel to and over the eighth rib, its median portion corresponding to the midscapular line. Rib exposed and intercostal vessels raised upward; a bone elevator placed under rib

and about 5 cm. removed with lion-jawed forceps. A similar portion was excised from rib above. Intercostal vessels ligated. Thoracic cavity opened, a quantity of chyloid fluid evacuated with force. Not all the fluid was removed. A large rubber tube (short) was anchored in the incision with silk. Incision was closed up to the tube with catgut. Antiseptic dressing.

For a time after the operation the patient did well, the temperature remaining normal. Beginning upon the tenth day the temperature became irregular, at times reaching 102° or 103°, and falling again to normal. The patient became dyspnœic and somewhat cyanotic with rapid pulse, which upon January 1st became uncountable. Upon January 3d the temperature rose to 103°, and the patient gradually sank, dying at 5.30 A.M.

The following is Dr. Kelly's report of his examination of the fluid removed from the left pleura December 8, 1904:

The fluid quite resembles milk in ordinary appearance—being whitish or slightly yellowish, opaque, and odorless. Its specific gravity is 1017. It contains a considerable amount of albumin—roughly, about 4 per cent. It does not respond to the ordinary tests for sugar—copper solutions, phenylhydrazin, and fermentation tests. The addition of ether followed by vigorous agitation causes the fluid to clear somewhat; the previous addition of a few drops of potassium hydroxide causes it to become much clearer. The addition of osmic acid causes the fluid to become quite blackish. On standing sometime a slight fibrinous deposit settled at the bottom of the vessel, and a small layer resembling the cream that forms upon milk formed at the surface. After standing in stoppered test-tubes and flasks at room temperature for ten days and two weeks, and being in the mean time several times exposed to the air, the fluid appeared quite "sweet."

Microscopically the fluid consists of cells (the character of which will be discussed presently) and a very large number of the finest granules; occasionally a single or several larger granules are observed, but almost all are very fine, even dust-like. They exhibit considerable molecular movement. The addition of a few drops of 1 per cent. osmic acid solution to the microscopic preparation causes the free granules, as well as similar granules in the under-described cells, to become darkish, blackish-gray in color. The addition of ether results in solution of the granules and their confluence to form larger masses, which become discolored, blackened, on the addition of osmic acid.

In addition to these granules, the fluid contains a moderate number of cells, a differential count of which reveals the following: lymphocytes, 76 per cent.; large mononuclear cells, 22 per cent.; polynuclear neutrophiles, 2 per cent.

The lymphocytes conform in all particulars to the lymphocytes of the circulating blood, and the polynuclear neutrophiles are also

readily recognized as identical with the predominating leukocyte of normal blood. The cells described as large mononuclear differ somewhat in appearance among themselves. A few of them resemble the lymphocytes, though they are larger; others recall the large mononuclear leukocyte of normal blood; but on account of the presence on the cells of granules resembling and behaving toward ether and osmic acid like the free granules of the fluid, of vacuolation, and of degenerative changes, it is impossible to say what some of these cells are. Other cells, however, are roundish in outline, vary somewhat in size, have a fairly large, usually eccentric nucleus; their protoplasm contains fatty granules and vacuoles; evidently they are desquamated endothelial cells.

Appropriate staining methods failed to reveal any bacteria in cover-slip preparations.

By culture methods a large unidentified, spore-bearing bacillus (probably contamination) was isolated.

The results of the examination of a second specimen of fluid received December 20th was identical with the foregoing (except that one portion was slightly pinkish, owing to the admixture of blood).

Almost daily examination of the blood (by day and by night) between December 8th and 20th failed to reveal filaria sanguinis hominis.

A specimen of the fluid was handed to Dr. Charles A. Fife, of the William Pepper Laboratory of Clinical Medicine, for chemical examination. He reports as follows: The fluid is faintly alkaline to litmus paper, but acid to phenolphthalein, requiring 20 c.c. of a one-tenth normal sodium hydroxide solution to neutralize 100 c.c. of the fluid. The specific gravity is 1016.4.[1] The fluid consists of 93.388 per cent. water and 6.61199 per cent. solids, of which 4.679 per cent. is albuminous (probably entirely serum albumin), 1.065 per cent. fats, and 0.5639 per cent. ash. The fluid after the removal of the fats and proteids reduces copper slightly, but does not respond to the phenylhydrazin sugar tests.

The following are the notes of the necropsy (No. 735), made by Dr. A. O. J. Kelly, January 3, 1905, six hours after death:

The body is that of a fairly well developed female subject with gracile bony development, slight musculature, and slight panniculus adiposus. The skin is pale; the nutrition poor. There is slight postmortem rigidity, but no postmortem lividity. In the right groin there is a linear scar (the result of an operation for femoral hernia). Posteriorly in the left thorax there is an opening into the pleura, a defect of a rib (due to resection), and a rubber tube draining the pleura. The preperitoneal adipose tissue is slight in amount. The thoracic and the abdominal viscera have their normal relations the

[1] Determined by a more sensitive instrument than was used in the other examination.

one to the other. The left pleura is non-adherent, except for a small area on the diaphragmatic surface; it contains 200 c.c. of rather thick "milky" fluid; in general it is pale, cloudy, and thickened, being in some places from 2 to 3 mm. thick; and it is covered (both the parietal and the visceral layers) with a rather thick or viscid, whitish, opaque, "milky" exudate. The left lung is small and compressed, and weighs 400 gm. It is mottled, grayish-black in color, of increased consistency, contains less air than normally, and reveals slight hypostatic congestion. The section surface is smooth, moist, and glistening, pale grayish in color, and exudes considerable frothy fluid. The lung crepitates throughout, and contains no tubercles and no cavities. The bronchi contain some serous or mucous fluid; their mucous membrane is pale, their calibre normal, but about them and extending somewhat into the adjacent tissue, there is apparently a slight amount of peribronchial connective-tissue overgrowth. The right pleura is dull, cloudy, pale, thickened, and universally adherent. As far down as the fourth rib the adhesions are firm and quite obliterate the pleural cavity. Below the fourth rib the adhesions are not so firm, and instead of obliterating the pleural sac, give rise to the formation of sacculi containing fluid; between the inferior surface of the lung and the diaphragm there is a large collection of fluid displacing the lung upward, but the lung extends down dome-like over the fluid—its edges reaching well down anteriorly, laterally, and posteriorly. The pleura contains a total of 550 c.c. of fluid, which, although somewhat "milky," is more correctly dull grayish in color, opaque, and "thinner" than the fluid obtained from the left side. The right lung weighs 650 gm. It is mottled grayish-black in color, of increased consistency, contains less air than normally, and reveals slight hypostatic congestion. The section surface is smooth, moist, and glistening, pale grayish in color, and exudes considerable frothy fluid. The lung crepitates through, and contains no tubercles and no cavities. The bronchi contain some serous or mucous fluid; their mucous membrane is pale, their calibre normal, but about them and extending somewhat into the adjacent tissue there is apparently a slight amount of peribronchial connective-tissue overgrowth. A few of the peribronchial lymphatic glands are quite small, firm, and anthracotic, and reveal no deviations from the normal; the great majority of them, however, are much enlarged, some of them very much enlarged, pinkish in color, and softened—almost diffluent. The pericardium contains 30 c.c. of clear, amber-colored fluid, and is non-adherent throughout. The heart is dilated, somewhat rounded in shape, and weighs 230 gm. It, more especially the right side, contains considerable dark clotted blood. The left auricle, the right ventricle, and the right auricle are dilated, especially the right auricle. The mitral orifice is contracted, and the leaflets thickened, indurated, and shortened. The chordæ tendinæ are also thickened and shortened.

The tricuspid orifice admits three fingers readily, and the valve leaflets are probably insufficient. The aortic and the pulmonary valves are normal. The left ventricle measures 8 to 13 mm. in thickness, the left auricle 2 to 4 mm., the right ventricle 5 to 8 mm., and the right auricle 1 to 2 mm. The heart is pale, brownish-red in color, and firm in consistency. The coronary arteries are normal. The arch of the aorta and the descending aorta are slightly thickened in patches. The œsophagus is normal. The omentum is normal in size and slightly fatty. The peritoneum is dull, cloudy, pale, and slightly thickened in places, especially the peritoneum of the anterior abdominal wall and that of the posterior peritoneal cavity toward the vertebræ near and extending into the pelvis. It is non-adherent throughout. It contains 1200 c.c. of grayish-white, opaque, "thin" fluid. The liver measure 27 x 18 x 5.5 cm., and weighs 1340 gm. It is normal in shape, of increased consistency, smooth on the surface, and has sharp edges. The section surface is smooth, glistening, and moist, mottled yellowish-brown (nutmeg) in color, and exudes an increased amount of blood. The gall-bladder is normal in size, contains fluid bile; the cystic, the hepatic, and the common bile-ducts are patulous. The spleen measures 11.5 x 6 x 2.5 cm., and weighs 90 gm. It is dark in color; the section surface is smooth; and the trabeculæ are slightly increased. The right kidney measures 10 x 5.5 x 3 cm., and weighs 130 gm. The capsule is slightly adherent, and when stripped leaves a slightly granular surface, dark in color. The kidney in general is of increased consistency. The cortex is 7 mm. in thickness. The left kidney measures 11 x 5.5 x 3 cm., and weighs 150 gm. It is similar to its fellow in appearance. Both ureters and both adrenals are normal. The urinary bladder and the external and the internal genitalia exhibit no deviations from the normal aside from the ovaries, which are slightly enlarged, whitish, firm, and cystic. The stomach is normal in size and position; its mucous membrane is slightly congested, especially on the posterior surface (postmortem). The entire intestinal tract appears normal. The pancreas is pale pinkish in color, quite firm, and exhibits no noteworthy deviations from the normal. The mesenteric glands are slightly enlarged, pinkish in color, and somewhat softened. The retroperitoneal glands, especially those just above the promontory of the sacrum, are much enlarged, quite red in color, and very soft, and many of them being quite diffluent; indeed, such is the softening and diffluence that it is impossible to distinguish gland from gland; higher up glands may be differentiated, but all are enlarged, softened, and red or pinkish in color. About the normal situation of the receptaculum chyli there are several enlarged glands scarcely to be differentiated from adjacent structures, but no receptaculum as a separate structure can be made out. Dissecting out the thoracic duct as high up as the clavicle no noteworthy change can be recognized—in particular

it is not enlarged or thickened. The brain and the spinal cord are not examined.

Anatomical Diagnosis. Sarcoma of the mesenteric, retroperitoneal, and peribronchial glands; bilateral pleuritis with chyliform exudation; chyliform ascites; mitral stenosis and insufficiency; dilatation of the heart; slight sclerosis of the aorta; passive congestion, œdema, and cyanotic induration of the lungs; passive congestion of the liver, spleen, and kidneys.

The following is the result of the microscopic examinations:

The enlarged mesenteric, retroperitoneal, and peribronchial glands are the seat of a new-growth that has almost entirely replaced or destroyed the lymphadenoid tissue—to a varying degree, however, in different glands. The new-growth consists of elongated, for the most part, spindle-shaped cells, with ovoid, rather vesicular nuclei, that stain well. The cells are arranged parallel or almost parallel the one to the other, but they are collected in fasciculi that course in varying directions, whence longitudinal, transverse, and oblique sections of the fasciculi are encountered. In many places the fasciculi seem limited to the sinuses, being apparently surrounded by a layer of flat endothelium; but in many places such arrangement and limitation are altogether wanting, and there is no appearance that warrants one in stating positively that the cells have been derived from proliferating endothelium. But little or no intercellular substance is apparent (by the ordinary methods of staining). Bloodvessels are comparatively few; those present have thin walls, mostly endothelium only, and they course amidst encircling fasciculi of tumor cells. Many of the retroperitoneal glands, especially those near the promontory of the sacrum, exhibit moderate necrosis. The other glands exhibit no retrograde alteration.

The pleura of the left lung is markedly thickened, the thickening involving the subendothelial connective tissue, the endothelium itself being wanting. This thickening consists of a highly vascularized fibrocellular tissue, the proportion of cells and fibrillar intercellular tissue varying in different regions. In general toward the free edge of the pleura the cellular collection consists of young fibroblasts and the ordinary round cells of inflammation; and the changes here merge rather rapidly into the more ordinary changes of fibrinous and fibrinopurulent exudation—a thick layer of which covers the pleura. The pleura of the right lung exhibits the usual lesions of adhesive pleuritis with considerable round-cell infiltration. Throughout both lungs there is an unusual thickening of the alveolar walls, which, however, is not uniformly distributed; in regions where it is less marked, there is considerable distention of the intra-alveolar capillaries, but where the thickening is more marked the capillary distention is obscured or absent. The thickening consists of a fibrocellular tissue rich in cells with elongated nuclei that stain well. Most of it appears to be white fibrous tissue, but

there is a considerable proportion of elastic tissue. In some places the cellular new-growth is excessive and completely obliterates the lumen of several adjacent alveoli. One receives the impression that here and there there is an unusual proliferation from one side of the wall of the alveolus into its lumen; in other regions, doubtless because of transverse section of such a polypoid growth, the new tissue reposes in or fills an alveolus without manifesting attachment to its wall. In addition to this connective-tissue overgrowth, many of the alveoli are more or less filled with an exudation consisting of leukocytes, desquamated epithelium, and a few erythrocytes. Many of the desquamated epithelial cells are excessively pigmented. This alveolar exudation is most marked just below the pleura of the left lung, where, together with the proliferation of the connective tissue of the alveolar walls, it gives rise to almost complete obliteration of the lumen of many alveoli.

The liver exhibits moderate dilatation of the central veins and adjacent capillaries, moderate pigmentation, and slight atrophy of the regional liver cells. The spleen exhibits considerable thickening of the trabeculæ, and considerable pigmentation—partly free and partly within the cells of the pulp. The kidneys exhibit moderate passive congestion and slight degeneration of the epithelium of the convoluted tubules. The uterus exhibits a slight grade of hyperplastic glandular endometritis. Otherwise there are no noteworthy microscopic alterations in the organs.

The fluid from the right pleural sac has a specific gravity of 1017. It contains 4 per cent. of albumin (Esbach). Microscopically there is considerable fibrin and a moderate number of cells, but none, or very few, of the fine granules observed in the fluid obtained by puncture from the left pleura during the life of the patient. A differential count of the cells reveals the following: lymphocytes, 58 per cent.; large mononuclear cells, 25 per cent.; polynuclear neutrophiles, 17 per cent. The lymphocytes and the polynuclear neutrophiles are readily identified. Some of the large mononuclear cells are doubtless degenerated lymphocytes, but the majority appear to be more or less degenerated and vacuolated endothelial cells.

The fluid obtained from the peritoneum has a specific gravity of 1016. It contains 3.5 per cent. of albumin (Esbach). Microscopically there is a moderate number of cells, and a large number of granules identical with those obtained from the left pleura. The granules are free and also contained within certain of the cells. They stain blackish with osmic acid, and are dissolved with ether. A differential count of the cells reveals the following: lymphocytes, 70 per cent.; large mononuclear cells, 29 per cent.; polynuclear neutrophiles, 1 per cent. The large mononuclear cells are similar to those found in the fluid from the left pleura, though they differ among themselves. Many of them contain fine granules that are

dissolved by ether, and stain black with osmic acid. They are doubtless desquamated endothelial cells.

Cultures from the right pleura and from the left pleura revealed staphylococcus pyogenes albus and aureus. Cultures from the peritoneal fluid revealed bacterium coli communis.

Within five hours of making the necropsy I inoculated three rabbits subcutaneously in the William Pepper Laboratory of Clinical Medicine with an emulsion of the softened retroperitoneal glands. One rabbit died the following day from peritonitis due to puncture of the intestine with the inoculating needle. The other two rabbits lived in excellent health, and were finally killed on April 6, 1905. No lesions were found at the necropsy.

REPORT OF A CASE OF METASTATIC ENDOTHELIOMA.

By Edward Jessup Wood, S.B., M.D.,

THE patient was a white man, aged forty-three years. His occupation was that of a salesman. There was nothing in his family history of any note except that his father died of carcinoma of the œsophagus, and a brother died of a complication of cardiac and renal affections. There was nothing in his previous medical or surgical history of any importance until the year 1900. During that year he noticed a small nodular mass on the surface of the right testicle. The testicle became painful and began to increase in size. It was firm in consistence and showed no sign of breaking down in any part of it. This condition gradually increased until the final removal of the testicle. In the opinion of the surgeon the growth was sarcomatous.

After a lapse of over two years, in 1902, the patient felt the presence in the remaining testicle of the same symptoms that formerly had attracted his attention—namely, the nodule of the size of a buckshot, the increase in consistence, the swelling, and the pain. Again he was advised to have the radical operation performed, which he consented to. In this instance the histological examination was made by a competent pathologist, who pronounced it a large round-cell sarcoma.

Following this second operation the patient's health improved, though he was never robust. In September, 1904, following a fall, he began to complain of backache. Seldom was he free from pain, which usually was of the dull, boring character, though at times it was of the stabbing variety. The patient thought that this more severe pain was relieved coincidentally with the voiding of his urine.

This pain persisted with intervals of comparative freedom followed by exacerbations which would necessitate his remaining in bed several days at a time. In many respects this pain simulated lumbago. During this time the patient kept his full weight and showed no signs of cachexia.

In January, 1905, he came under my care with the simple history of pain in the lumbar region. This pain was most obstinate and resisted the largest doses of morphine. About the middle of January I detected in the left upper quadrant of the abdomen, just within and below the splenic area, a globular-shaped mass which was absolutely immovable in any position the patient might assume. This mass was uncovered by gut and was perfectly flat on percussion. The spleen was normal in size and position. The left kidney could not be palpated in its normal position, while the right was palpable. All other viscera were normal to physical examination.

The provisional diagnosis of malignant disease of the left kidney was made and it was advised that an exploratory incision be made with a view to a possible nephrectomy. The exploratory incision was made and revealed directly beneath the peritoneum a kidney enlarged to about three times the normal size, being irregular in contour and consistence, which was placed almost transversely across the abdomen, corresponding to a line drawn from the gall-bladder to the spleen. The organ was bound down to the lumbar spine by very dense adhesions, and could not be moved a fraction of an inch. It transmitted the pulsations of the abdominal aorta. The surgeons found a nephrectomy to be impracticable and the wound was closed with nothing done. He rallied feebly from the shock of even this slight operation.

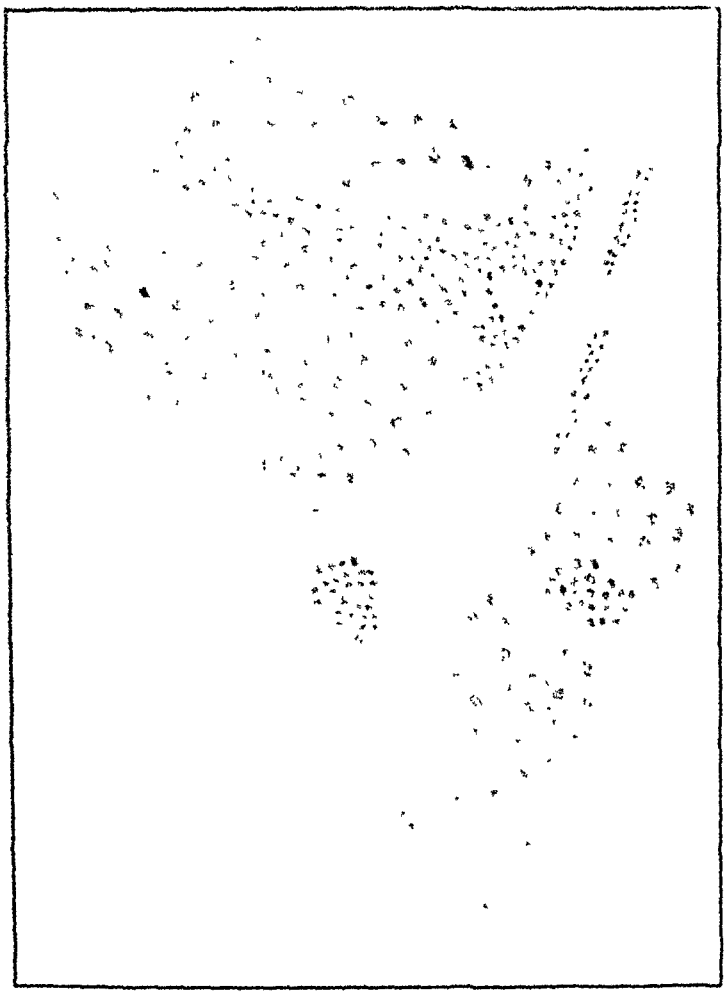
A few days later it was noticed that the right leg was considerably œdematous and there was a marked difference in the temperature between it and the left leg, which was much warmer. It gave the patient considerable discomfort, though not acutely painful. The œdema soon extended to the scrotum.

For a few days the patient was free from the lumbar pain, but it soon returned with added violence and could not be relieved by morphine.

Ten days after the operation the patient was seized with pain in the side, over the base of the right lung and became cyanosed. For a short time his condition was grave, but he rallied. Just previous to this he had begun a "prune-juice" expectoration, which now became very fetid and was expelled by a painful cut-off pleuritic cough. The temperature, which hitherto had been normal, now rose to 104°, and delirium developed. It was apparent that the condition was gangrene of the lung. Twenty-four hours later the patient died with a large pulmonary hemorrhage.

An autopsy was immediately done. The tumor mass was found to be the left kidney. It was bound to the spine by such dense

adhesions that it could only be torn out piecemeal. There was nothing that resembled normal kidney tissue and, in fact, it was structureless save in small areas, the bulk being made up of a mass of soft caseous material. At one point it was broken down. The right kidney was normal excepting a slight increase in size which was probably compensatory hypertrophy.



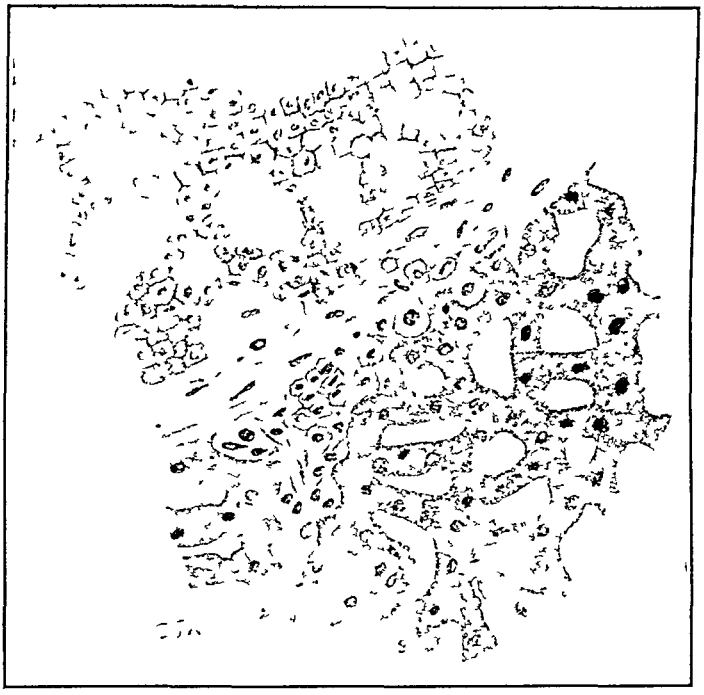
Macroscopic examination of the liver showed no changes except that on the anterior surface there was a small round nodule, white in color, depressed in the centre, and sharply defined.

Between the right lung and the costal pleura there was found some delicate adhesions, which were easily broken up. The base was consolidated and showed a small area of gangrene. No other gross changes were found in any of the remaining organs.

The left kidney was so diseased that it was with some difficulty a suitable specimen could be obtained for the microscopic examina-

tion. The sections were stained with hæmatoxylon and eosin. Only small portions of the tissue here and there would take the stain, the bulk of the section being made up of large homogeneous areas apparently of the type of hyaline degeneration. Occasionally this degenerated material had the appearance of coarse connective tissue, sometimes of the fibrous variety, and again it had a peculiar appearance resembling degenerated muscle fibre. Fig. 1 represents a section taken from the most organized part of the left or diseased kidney. The large T-shaped mass of connective tissue is composed of what appears to be degenerated fibrous tissue, which is homo-

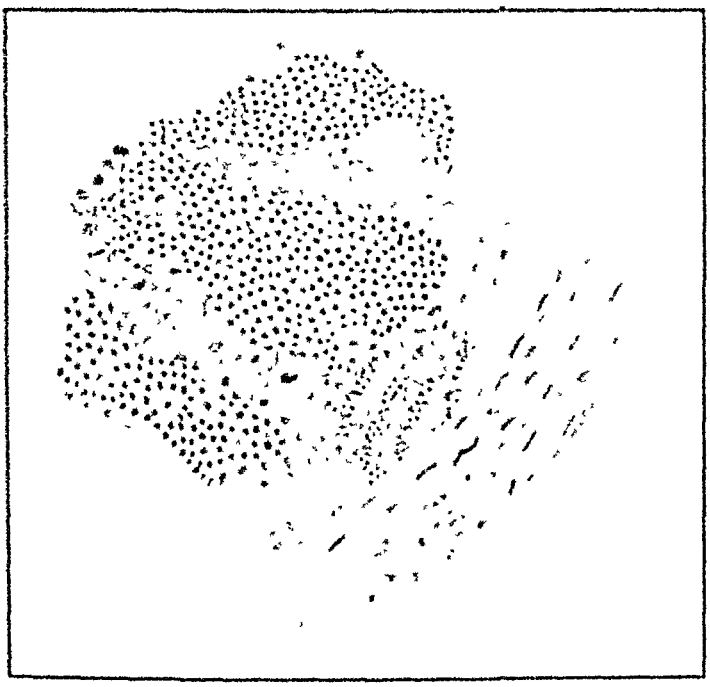
FIG 2



geneous in appearance. Interspersed throughout the fibres are found accumulations of small round cells which take a deep stain with hæmatoxylon. The cells have the appearance of round-cell sarcoma. In smaller numbers is another cell not so well organized which is but little affected by the stain. The greater portion of the section is made up of large cells, usually polygonal in shape, with large nuclei which take a deep stain with hæmatoxylon. The cells are packed closely together and often it is difficult to make out the outline of the individual cell. The protoplasm of the cells is homogeneous and stains but faintly. Between these cells is seen an extensive mass of the same small round cells described above as

occurring in the connective tissue. In addition to this large mass of cells there are several smaller pockets. The large cells are evidently of the endothelial variety. These large cells with occasional small round cells were found here and there in small clusters throughout the hyaline mass of tissue.

Sections of the liver through the small nodule above mentioned were also stained in eosin and hæmatoxylon. The liver cells are found in the majority of instances to be practically normal except near the surface of the organ, where there are few evidences of a fatty degeneration, but this is extremely limited and only found where the endothelial growth is beginning to encroach on the liver tissue. Fig. 2 shows the section of liver in which the capsule is



thickened and beyond the capsule, but in intimate contact with it is a mass of endothelial cells. This mass of cells makes up the nodule which was described above. These endothelial cells are placed around large irregularly shaped spaces, making a sort of mesh work with no particular pattern. In the sketch the capsule of the liver shows invasion of these endothelial cells. In the liver tissue is a cleft which is filled with connective tissue and with the same endothelial cells. The shapes of the cells vary and apparently the cells adapt their shapes to these open spaces. The cell outline is indistinct. The protoplasm of the cell stains faintly and is homogeneous. The nuclei of the cells are large, well defined, and take a deep stain. The cells closely resemble in many ways the cells found in the kidney; the chief differences are in shape and size.

There is a marked absence of the small round cells which are so abundant in the kidney. There is, however, a cell somewhat larger but otherwise much resembling it which had invaded the liver tissue.

Evidently from the general condition of the organ the growth had made but little headway in the liver. Had the little nodule not have attracted attention there would have been nothing in the general appearance of the organ to suggest a metastasis. The liver was otherwise normal to gross appearances.

The lung tissue showed hemorrhagic infraction. It becomes a question to account for this condition of infarction. Either it was due to an infectious embolus or else to the invasion of the tumor cells. In the light of an absence of any active or healed tuberculous lesion and no previous history of any pleuritic trouble it was natural to suspect a pleuritic metastasis, especially when the frequency of this site for endothelioma was considered. Fig. 3 shows a section through the diseased lung with the pleura. Just within the pleura is noted the same cell which predominated Figs. 1 and 2. This cell is in intimate contact with and within the bloodvessels, and this is the only tissue which demonstrates this relation to the vascular system. There is an extravasation of blood throughout the tissue, being a part of a general condition. What role the tumor cells played in the fatal occlusion of a bloodvessel is a question. Certainly the lung was invaded by the tumor before the fatal accident occurred. It is likewise certain that there was in the body of the patient a source for the production of an infectious embolus. The lung was probably the last organ invaded, and yet in many respects it gave the most typical demonstration of endothelioma. In many places the endothelial cells so arranged themselves that it gave the appearance of a tubular gland. None of these cells were discovered in the pleura, although it showed signs of marked changes but more of an inflammatory nature.

The tumor of the testicle was unquestionably a large round-cell sarcoma, and it is altogether likely that this was the original source of the disease. If this be the case and the metastatic tumors being of the endothelial variety, it is another evidence that endothelioma is a pure sarcomatous tumor.

It is a notable fact that the metastasis was exceedingly slow for a sarcomatous tumor. From the time of invasion to death there elapsed a period of nearly five years.

I am indebted to Dr. C. H. Bunting, of the Johns Hopkins Hospital, for valuable assistance in the microscopic work.

THE EXCRETION OF ACETONE AND DIACETIC ACID IN A CASE OF PERNICIOUS VOMITING OF PREGNANCY.

By Helen Baldwin, M.D.,
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(From the Laboratory of Dr. C. A. Herter, New York.)

The degeneration in the liver noted by Ewing[1] in cases of pernicious vomiting of pregnancy, as well as in eclampsia, suggested to me that in these conditions there might be found urinary changes due to the breaking down of the proteid molecule and to the associated disturbance of function of the liver cells. The histological alterations in these cases are similar to those in poisoning by phosphorus, where the urine is known to contain pathological organic acids and acetone. It is known also that in phosphorus poisoning the oxidation of the body as expressed by the intake and output of oxygen and by the excretion of urea is greatly diminished. Wakeman[2] has recently shown that in phosphorus poisoning the yield of hexon bases from the proteids of the liver is distinctly diminished, especially in the case of arginin, and it is likely that the damage to the proteid molecule during life (which the changes represent) is related to the diminished oxidations just mentioned.

The similarity of the hepatic lesions seen in phosphorus poisoning and in a late stage of pernicious vomiting of pregnancy thus points to the likelihood that the chemical alterations in the cells are allied and are attended by similar disorders in metabolism. Leucin and tyrosin (products of proteid cleavage) have been found in the urine in both conditions, and are probably present in the livers in cases of pernicious vomiting as well as those of phosphorus poisoning. The following case shows that in the vomiting of pregnancy acetone may be associated in the urine with at least one organic acid, namely, diacetic. Unfortunately, it was not practicable in this case to thoroughly search for leucin, tyrosin, and other amido acids.

This case is interesting not only on account of the finding of these acetone bodies, but also from the evidences which it gives of a high degree of intestinal putrefaction, by the excretion in the urine of large amounts of phenol, indol, and combined sulphuric acid.

The patient was in the care of Dr. Lotta W. Myers, who has kindly allowed me to publish her notes of the case. The history is as follows:

The patient is twenty-six years old. She has never had any

[1] The Pathological Anatomy and Pathogenesis of the Toxæmia of Pregnancy, American Journal of Obstetrics, 1905, vol. li. p. 145.

[2] Zeitschr. f. physiolog. Chemie, 1905, xlix. p. 335.

serious illness. She has always had very severe occipital headaches at the time of her menstrual periods, but has not had attacks of vomiting at that time. She has all her life been very constipated, and states that her stools have always been white; there has not, however, been any peculiarity in the color of her bowel movements during the time that she has been under observation. She was married in November, 1904. The last menstrual period was on January 8, 1905. She began to have nausea about February 5th, but did not have any attack of vomiting until February 20th. At this time she was very constipated. During a long journey in the cars she had a violent attack of vomiting. This was relieved by ordinary treatment, with rest and restricted diet, but on March 4th another very violent attack of nausea and vomiting occurred.

From March 8th to March 30th the patient was kept in bed. The feeding during this time was almost entirely by nutrient enemata. Whenever an attempt was made to begin feeding by mouth a very severe attack of headache, nausea, and vomiting would occur. She had a good deal of pain in the back and of intestinal colic, with flatulence. She had at times great restlessness, with twitching in the limbs, and blurred vision. On March 9th, 10th, and 11th she had four small hemorrhages from the bowels, probably from the rectum.

On March 15th the patient had another severe attack of vomiting. She could not retain any food, and vomited ten times during the day. On March 20th she vomited fourteen times during the day. Her condition became progressively worse. Each attack of vomiting became more alarming than the previous one. On March 30th an operation seemed imperative. She was removed to the Sloan Maternity and an abortion performed by Dr. Cragin. After this the patient made an uninterrupted recovery. The following are the urinary reports before and after the operation:

Urinalysis. March 25, 1905. Volume, 600 c.c.; specific gravity, 1028; reaction acid; urea, 8.86 grams; trace of albumin; no sugar; no leucin; no tyrosin; marked reaction for acetone and diacetic acid; a few epithelial cells and calcium oxalate crystals in the sediment; no casts; the urine was acidified with sulphuric acid and shaken out for organic acids; the organic acid thus obtained from the twenty-four-hour specimen was equivalent to 12.2 c.c. $\frac{N}{10}$ NaOH; indican and phenol both were present in large amounts.

29th. Volume, 640 c.c.; clear, light colored; reaction, acid; urea, 8.812 grams; total nitrogen, 4.112 grams; nitrogen of ammonia, 0.1954 grams = 4.75 per cent. of total nitrogen; marked reaction for acetone and diacetic acid; marked reaction for indican and phenol; combined sulphuric acid 0.1820; preformed sulphuric acid 0.3531; ratio $\frac{H_2SO_4 \text{ preformed}}{H_2SO_4 \text{ combined}} = 1.9$. Bladder epithelium, a few

leukocytes, and a few calcium oxalate crystals in the sediment; no casts.

30*th*. Twenty-four hours before operation. Volume, 800 c.c.; color, light amber; reaction for diacetic acid more marked than on previous day; large amount of acetone.[1]

31*st*. First day after operation. Volume, 1260 c.c.; distinct reaction for albumin; granular casts; diacetic acid and acetone both present in considerable amount, but the reaction is not as marked as before the operation; large amount of phenol.

April 1st. Volume, 1860 c.c.; distinct reaction for acetone; trace of diacetic acid; phenol present, but not as much as before operation.

2*d*. Volume, 1260 c.c.; specific gravity, 1015; no albumin; marked reaction for diacetic acid and acetone; large amount of phenol and indican.

3*d*, 4*th*, and 5*th*. No acetone; no diacetic acid; marked reaction for phenol.

7*th*. Volume, 1080 c.c.; specific gravity, 1025; no albumin; diacetic acid present; trace of acetone; phenol, distinct trace.

8*th*. Volume, 1260 c.c.; specific gravity, 1015; very marked reaction for diacetic acid; trace of acetone; phenol, distinct reaction, but not as much as before. *The patient had a severe headache, with nausea and vomiting on this day.*

9*th*. Volume, 1380 c.c.; specific gravity, 1012; no albumin; distinct reaction for diacetic acid, but not as much as on April 8th; faint trace of acetone; distinct reaction for phenol; preformed sulphuric acid 0.4751; combined sulphuric acid 0.1160.

$$\text{Ratio, } \frac{\text{preformed sulphuric acid}}{\text{combined sulphuric acid}} = 4.0.$$

The patient improved rapidly from this time. No further examination of the urine was made until April 27th, when the report was as follows:

27*th*. Volume, 1000 c.c.; no albumin; specific gravity, 1016; no diacetic acid; faintest trace of acetone; trace of phenol; marked reaction for indican; no casts; bladder epithelium and a few leukocytes in sediment.

28*th*. Volume, 1000 c.c.; specific gravity, 1028; no albumin; no acetone; no diacetic acid; distinct trace of phenol, large amount of indican.

29*th*. Volume, 1000 c.c.; specific gravity, 1018; no albumin; no diacetic acid; faintest trace of acetone; trace of phenol; large

[1] The method used in testing for acetone was as follows: 5 c.c. of strong H_2SO_4 were added to 100 c.c. of urine and distilled. About 60 c.c of the distillate were collected. A portion of this was warmed in a test tube and rendered alkaline by the addition of a few drops of a solution of potassium hydroxide. Then Lugol's solution of iodine was added drop by drop until there was a slight excess. Then the solution of potassium hydroxide was again added, drop by drop, until the brown color disappeared. With this test, if acetone is present, there is a prompt precipitation of iodoform.

amount of indican; preformed sulphuric acid 0.5444; combined sulphuric acid 0,1008; ratio, $\frac{\text{preformed sulphuric acid}}{\text{combined sulphuric acid}} = 5.4$.

In reviewing the points of interest in this case the most important observation is in the finding of the large amounts of acetone and diacetic acid in the urine. The diacetic acid which was present in large quantities in the specimen of March 25th had increased in the few days before the operation.

While the presence of acetone is not of so much significance as that of diacetic acid, its persistence and increase would indicate that the toxæmia was becoming more grave. The promptness with which the diacetic acid and acetone disappeared from the urine after the operation, and their temporary reappearance after eight days, when the patient had a severe attack of headache and vomiting, are also noteworthy.

The degree of intestinal putrefaction is very striking, as has been noted by others in cases of this kind. Indican and phenol are both present in the urine in large amounts, and the ratio of the combined (aromatic) sulphates to the preformed sulphates, that of 1 to 1.9, indicates an extreme degree of bacterial activity in the intestines.

While the report of a single case is of relatively little value, it has seemed well to publish this record as emphasizing the importance of testing for acetone and diacetic acid in the urine of similar patients.

A PROCEDURE FOR OPENING THE PERICARDIUM.

By J. H. Bacon, M.D.,
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It is a well-attested fact that pericarditis is recognized much more frequently at the postmortem examination than during life. That it is difficult to recognize no one will gainsay, as many of the cases develop during the last hours of life as complications of some other diseases; then, some cases which might be recognized are overlooked because the patient is too ill to justify a thorough physical examination. In 4700 cases of the medical service of the Lakeside Hospital there have been diagnosed some 45 cases of pericarditis, of which 13 were fatal—a mortality percentage of 29. In the same service there were 245 cases of pneumonia, with a mortality of 29 per cent., while in 15 cases complicated by pericarditis there was a mortality of 71 per cent., an increase of over 40 per cent. in the complicated cases. There were 158 cases of acute articular rheumatism treated, with a death rate of $3\frac{1}{3}$ per cent.; while in 9

cases complicated by pericarditis mortality was 30 per cent. This shows from the clinical side what a marked increase of deaths occur in the two diseases with which pericarditis is most commonly associated, if they are complicated by it.

It may seem that one who has been working along pathological lines is afield in developing an operation, but that is a logical origin. My experience at the autopsy table has impressed on me three points in regard to pericarditis: (1) its great frequency as a complication of other diseases; (2) the comparative ease with which the pericardium may be explored through an opening in the sternum; (3) the necessity of local treatment in severe cases. It seems to me that if advantage is taken of the anatomical relation of the pericardium to the posterior surface of the sternum, there is afforded an easy route of attacking the pericardium and applying local treatment in suitable cases.

The treatment of the pericardium should be the same as that of other serous cavities—viz., joints. In simple conditions, rest and local application of ice-bag or heat. When there is no reaction to this treatment, it is aspirated when there is simple effusion, and opened when pus is present. These procedures should also be applied to the pericardium in similar cases.

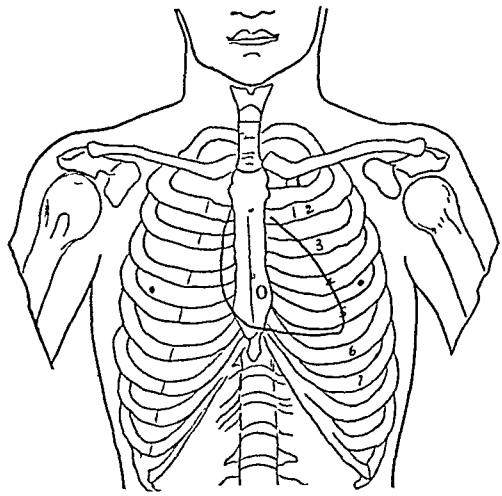
I have been impressed, as I lifted the sternum after severing the ribs on either side during postmortem examination, by the broad connection of the posterior surface of the lower one-third of the sternum with the anterior surface of the pericardium. The pleuræ form the lateral boundaries to this tissue of the anterior mediastinum. This forms, what it seems to me, a feasible route for attacking the pericardium, as it has a correct anatomical basis. A layer of prepericardial fat, which sometimes contains lymphatic glands, normally separates the sternum and pericardium.

In some pathological conditions, as with adherent obliterative pericarditis, fibrous tissue invades this fatty tissue, and then the pericardium is bound to the sternum. The size and shape of this area vary. Voinitch and Sionojensky have worked out a composite picture of the cases in their experience. It forms a triangle, with the base lying to the left of the midline and opposite the fifth intercostal space, and the apex extending down the fifth intercostal space. While I have not worked out a composite picture, I have found but a single case in 110 autopsies that has failed to have a connection that one would miss by going through the sternum at this point. This was in a child aged eight years, with a pigeon-shaped breast. Using this connection as a basis, I have developed the following procedure for entering the pericardium.

A skin incision is made parallel to the mid-line of the sternum and ½ cm. to its left, extending from the level of the upper border of the fifth costal cartilage to the lower border of the sixth. This incision is deepened until the sternum is exposed. This is laid

bare over an area of 2.5 cm. and the periosteum is raised. With a trephine, 2 cm. in diameter, a circular window is made through the bone in the same manner as in trephining the skull. One opens upon the anterior mediastinum and the pre-pericardial fat then comes to view. The fat is dissected away and the anterior surface of the parietal pericardium is exposed. The pericardium is raised by two mouse-toothed forceps, and an incision is made between them. From this opening into the pericardium, which is at its lowest level, just above its union with the diaphragm, one can readily explore the pericardial cavity, and then institute the treatment that is necessary, according to the conditions present. In children the sternum is narrower, and one may make

FIG 1



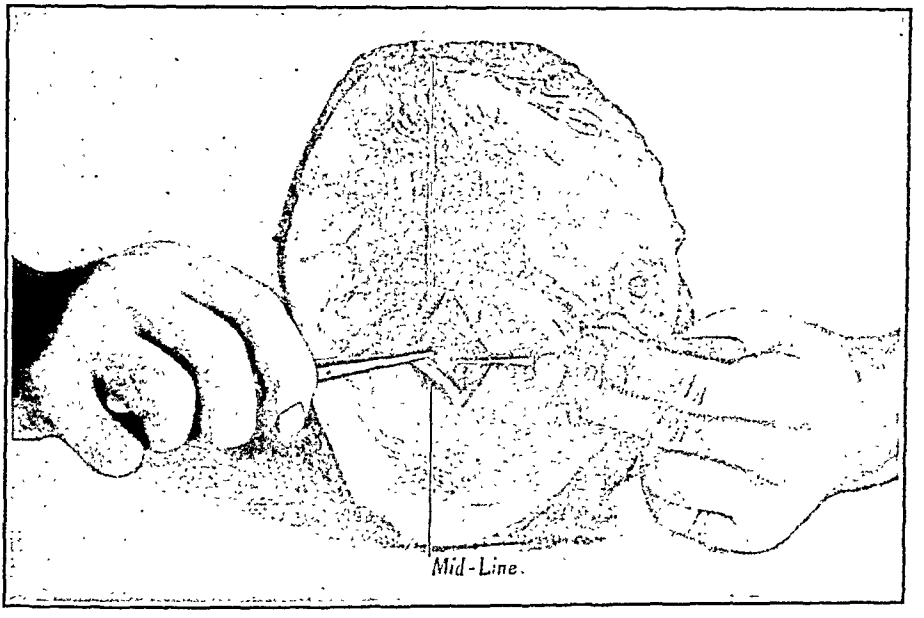
Position of pericardium and window in sternum.

the incision nearer the midline, so that the trephine opening will not separate a rib from the sternum. I have found one case in which the sternum, in the adult, was just 2.5 cm. in width. The internal mammary artery lies from $\frac{1}{2}$ to 2 cm. beyond the sternum, and so it is not injured. The sternum averages about $\frac{3}{4}$ cm. in thickness in the adult, but varies considerably. I have had one case, and have seen several, in which there was an opening in the sternum at the level of the fifth intercostal space, about $\frac{3}{4}$ cm. in diameter. This condition is due to a developmental error (lack of fusion of two sides in the embryo).

After one has trephined it is best in those cases where there is pus to fill the cellular portion of the cut edge of the bone with a 15 per cent. iodoform wax, to guard against infecting the bone and to aid in stopping (any excess of) hemorrhage. When the anterior mediastinum is entered one may find some enlarged lymph nodes. I have

found some as large as lima-beans in cases of pneumonia. These should be excised with the fat. When the pericardium is bared it can be identified by feeling the heart beating, and its convexity will be caudal-ward. In those cases where the diaphragm is raised above the normal position by pressure from below, the convexity will be cephal-ward. The pleuræ may be distinguished on either side by the color. In purulent cases it is well to place a purse-string suture before incising the pericardium. A rubber catheter is inserted to aid in flushing and should be left for drainage. Gauze should be packed around the catheter to protect the anterior mediastinum. Better drainage will be afforded if the patient is

FIG. 2



Photograph of specimen. Pericardium held by forceps.

turned on the right side. (The after-treatment will depend on conditions found and their reaction to drainage and flushing with warm saline.)

The advantages presented by this procedure:

1. It is anatomically correct.
2. Easy approach to the pericardium by definite landmarks that are readily made out.
3. The best possible drainage afforded.
4. It permits of ready inspection of the heart and the pericardial cavity.
5. It is devoid of mechanical danger, such as hemorrhage and rupture of the pleura.
6. It can be done under gas anæsthesia.
7. It requires but five to fifteen minutes for its completion.

8. No after-deformity.

9. Least possible danger of spreading infection to the pleural cavities or to the peritoneum.

The advantages and the relative simplicity of the operation are such that the opening of the pericardium should become more frequent. In the height of our present knowledge it seems conservative to treat all cases of purulent pericarditis and large serous effusions, and those cases of fibrinous pericarditis not reacting to rest and the ice-bag by drainage. Cases should be operated upon earlier in the disease before myocarditis has developed from absorption of toxins.

I have performed this operation on eighteen autopsy subjects. Once on a dog, in which I infected the pericardium, manipulated the heart roughly, and simply left drainage to see what would result. Estimated hemorrhage was 5 c.c. The dog was quite sick for five days, then rapidly improved. In two weeks only a healing wound remained. I have made some recent observations on the position of the heart in effusion and the relations of the pericardium to the thorax while under tension, which will be reported later.

THE PERMEABILITY TO BACTERIA OF SURGEONS' RUBBER GLOVES.

By Herbert Fox, M.D.,

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AND

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Following a discussion among surgeons as to the absolute impermeability to bacteria of the rubber gloves so generally used in modern surgery, the writers determined to undertake a series of experiments which should determine conclusively whether or not the rubber glove was an absolute safeguard against the implantation of bacteria from the hand of the surgeon to the field of operation.

The fact that pure rubber tissue is impassable to bacteria was not questioned, the doubt hinging rather upon the process of manufacture of the gloves, it being obvious that fissures, minute perforations, or areas of extreme thinness might not infrequently occur, and would of necessity be a prolific source of wound contamination.

Six pairs of gloves of standard make were selected at random from a lot purchased for hospital use, and were subjected to the following experiments:

No. I. The fingers of the gloves were cut off at the web and placed in test-tubes previously filled with peptone broth, high enough for the finger to be immersed about one inch when also filled with bouillon. The free cut edge of the glove finger was tied about the rim of the test-tube, the mouth plugged with raw cotton within the rubber finger, and sterilized in this arrangement for three successive days in the Arnold steam sterilizer. Upon the expiration of the proper sterilization the tubes were separated into two sets of three tubes and one control each. After carefully removing the cotton stopper two tubes were inoculated with the B. coli communis, two with staphylococcus pyogenes albus, and two with streptococcus pyogenes, the control placed under the same conditions, but not inoculated.

After twenty-four hours' growth at 37° C., one tube of each culture was plated by the stroke method from the bouillon without the glove finger. Extreme care was taken in removing the finger that no culture within the rubber should escape. The bouillon in the tubes that had been in contact with the glove finger containing the B. coli communis and staphylococcus pyogenes albus gave a positive culture of the corresponding organism, but not any culture appeared on the tube containing streptococcus pyogenes. There was a possible source of contamination in the two tubes which gave cultures, for, when taken from the incubator, the rubber that had been inverted over the rim of the test-tubes was found loose; it was moist when tied, and dry when it came from the incubator. The same conditions prevailed in those cultures allowed forty-eight hours' growth—*i. e.*, the streptococcus pyogenes negative, staphylococcus and B. coli communis positive. The controls consistently were sterile. If the growth was due to contamination or faulty technique, the reasons for the streptococcus pyogenes' failure to grow were probably incorrect temperature for this particular strain of organism, or unfavorable reaction of the medium. The growth of the second twenty-four hours was at room temperature.

As this was obviously a delicate and difficult method, it was dropped. When the next experiment was done there was no satisfactory strain of streptococcus pyogenes in the laboratory, so the work was continued with the other two organisms.

No. II. This time whole gloves were used, immersed in large flasks of bouillon. The gloves were placed in bouillon about halfway to the wrist, and bouillon poured into three to the same height. The gauntlet was reflected over the rim of the flask and tied tightly. To avoid any risk of external infection a beaker was inverted over the cotton stopper. It may be said here that it is expedient to grease the glove at the surface that comes in contact with the cotton, otherwise the rubber will pull up when the stopper is removed.

After sterilization, as before, the bouillon within the gloves was inoculated with the B. coli communis and staphylococcus pyogenes

albus and allowed to grow forty-eight hours, when cultures were made from within and without the gloves; the former resulted in a pure culture of the germ, the latter being sterile. This experiment was repeated with like results.

No. III. The last test was severe. The flasks and gloves were prepared, stoppered, covered, and sterilized as before, but at the time of inoculation the wrist of each glove was twisted in order to distend the glove, making the pressure greater than when on the operator's hand; then it was tied tightly and the flasks were put at 37° C. for forty-eight hours. The cultures made from the bouillon showed no B. coli communis or staphylococcus pyogenes albus. Entirely new gloves were used each time.

The results of these experiments were such as to justify the writers in arriving at the conclusion that rubber gloves are absolute barriers against wound infection by the hands of the surgeon, in so far that they are absolutely bacteria proof. Any infection occurring must be introduced into the wound from some point not protected by a glove.

THE EFFECT OF EXPERIMENTAL CONDITIONS ON THE VASCULAR LESIONS PRODUCED BY ADRENALIN.

By Leo Loeb, M.D.,

AND

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OF PHILADELPHIA.

(From the Pathological Laboratory of the University of Pennsylvania.)

The pathogenesis of diseases of the bloodvessel walls has been one of the most obscure subjects in pathology, partly because we have been entirely dependent on clinical and anatomical observation for our knowledge of the process; partly because arteriosclerosis and atheroma are found associated with such varied pathological and clinical conditions that it is almost impossible to determine which of these stand in an etiological relation. The experimental production of such changes in the bloodvessel walls is therefore a noteworthy advance in our study of the subject, and the discovery by Crocq and Gilbert and Lion that the injection of bacteria and their toxins may produce such changes was of very great interest. Two years ago the subject was brought into even greater prominence by Josué's discovery that the repeated injection of small amounts of adrenalin into the veins of rabbits produced changes in the aorta which have a close resemblance to atheroma, even calcification being not infrequent. The results obtained by Josué were confirmed by the observations of Rzentkowski, Erb,

Jr., Fischer, Gouget, Pic and Bonamour and others. Experience showed, however, that lesions did not always follow the injections and that the results were influenced by certain unknown factors.

It was with the hope of throwing more light on this obscure subject that the experiments described in the present article were undertaken, the influence of a number of conditions which are supposed to bear a relation to atheroma being studied. Furthermore, this experimental method of producing lesions in the bloodvessels will provide us with means of determining accurately the effect of certain substances believed to be curative in arteriosclerosis of man.

Our experiments may be divided into three groups, as follows, each group being accompanied by a number of control rabbits to which only injections of adrenalin were given.

1. Some experiments were made to determine the effect of excision of the thyroid gland, which was of interest not only because von Eiselsberg has shown that calcification of the aorta may follow this operation in goats and sheep, but also because thyroidectomy seems to have some effect on calcium metabolism and on the healing of fractures, and therefore might have some influence on calcification of the aorta.

2. The influence of lesions of the kidney was worthy of study because nephritis is regarded as one of the most important causes of arteriosclerosis in man, and because interference with the excretion of adrenalin might increase its toxic action. For this reason bichromate of potassium was administered to a series of rabbits in order to produce nephritis; and in a second series one ureter was ligated—this operation not only causing temporary mechanical interference with secretion, but also (as is believed by some investigators) the production of nephrotoxins.[1]

3. As it might be supposed that the injections would have an influence on the structure of developing bloodvessels, and the administration to pregnant animals would also show whether the injections interfered with the course of pregnancy, or whether the latter influenced the toxic action of adrenalin, several pregnant animals were made objects of study.

4. Other questions came up for study during the course of experiments. These were, whether other lesions or results of adrenalin injections had any relation to its toxic action on the aorta, and whether the duration of the experiment, the number of injections, or the amount of adrenalin injected had most influence on the final result.

It has been maintained, especially by Huchard, that increase of blood pressure is one of the most important factors in the production of atheroma, and the thought naturally suggested itself

[1] This view can at present be regarded only as an hypothesis.

that the structural changes following the injection of adrenalin might be connected with its blood-pressure raising property. This led certain investigators to make experiments in this direction. Josué found that nicotin, which causes a rise of pressure, has no effect on the structure of the bloodvessel walls; and L. Braun found that the injection of amyl nitrite with the adrenalin prevents the rise of pressure, but does not interfere with the production of vascular lesions.

Adrenalin chemically seems to be made up of an hydroxyethylamine radical united with a base of orthodioxy benzine. The latter is known as pyrocatechin. Adrenalon, the keton of adrenalin, has been obtained synthetically through the interaction of methylamin and chloracetylpyrocatechin.

As recent studies of Dakin have shown that intravenous injections of 0.002 gram of pyrocatechin cause marked rise in blood pressure, and Baum has shown that its action on the bloodvessels of the skin is similar to that of adrenalin, it seemed worth while to determine whether it has also the power to produce lesions in the aorta. We therefore made a series of experiments in this connection.

1. INFLUENCE OF THYROIDECTOMY. The thyroid glands were removed from four rabbits. In three of these the parathyroids were certainly removed also, and as no trace of these could be found in the other (No. 2) at autopsy, we may feel fairly sure that they were removed in this also.

After our experiments had been planned an article by Lortat-Jakob and Sabarianu appeared in which it was stated that after complete thyroidectomy injections of adrenalin produced no changes in the aorta.

Our experiments did not confirm this idea. Only one of the four (No. 3) showed no sclerotic lesions; two of the others showed slight alteration; and in the fourth (No. 2) the entire thoracic aorta was converted into a calcareous tube with a wall like that of the trachea. The three positive results show clearly that removal of the thyroid glands does not prevent the toxic action of adrenalin on the aorta, although it cannot be said that they show that the effect is more pronounced.

2. INFLUENCE OF RENAL LESIONS. Potassium chromate was administered by stomach tube to twelve rabbits, in quantities varying from 7 to 40 c.c. of a 1 per cent. solution, over periods ranging from ten to forty days. In all cases the urine was tested at frequent intervals and found to be constantly albuminous. In two of these (Nos. 2 and 22) the chromate was administered during the latter part of the experiment only. In four other rabbits the left ureter was tied close to the kidney, and at autopsy occlusion was found to be complete in each case. In the latter injections of adrenalin were given twice a week until death, which occurred from twenty-

two to forty-seven days after operation. The only one (No. 5) which showed any aortic lesion lived twenty-seven days.

On the whole, the lesions were no more severe in these rabbits than in corresponding controls, which seems to show that interference with the elimination of adrenalin does not increase its toxic action. This agrees with the results obtained by Embden and v. Fürth; according to the latter investigators the blood-pressure raising power of adrenalin is quickly destroyed in the blood, and it also agrees with the fact shown by Cybulski that its effect on blood pressure was as transitory after double nephrectomy as before.

3. INFLUENCE OF PREGNANCY. Six rabbits were treated during the whole, or almost the whole, of pregnancy—some almost every day. In three of these no alteration of any sort was found in the aorta; in one of them there was a small spot of certain change; in one slight and doubtful lesions were found, and in the last these were fairly marked, but as injections were continued about four weeks after delivery this case must be excluded when we are considering the influence of pregnancy on the lesions produced by injections of adrenalin.

It was evident that the course of pregnancy was not influenced, and the young, which were seen in three cases, appeared to be quite healthy. Several of the offspring from two births were dissected, but no lesion of the arteries or other abnormality was to be found.

We may therefore state: 1. Adrenalin injections do not interfere with the course of pregnancy or delivery in rabbits. 2. They have no effect on the development of the vascular system of the fetus. 3. It seems probable that pregnant animals are less susceptible to the toxic action than are others. The latter conclusion cannot yet, however, be regarded as a definite one.

4. RELATION OF CERTAIN OTHER LESIONS. In some rabbits aortic changes occur after a very short time and from very small amounts of adrenalin. Thus, Rzentkowski reports lesions after four days, and only a little more than 0.5 c.c. of the 1:1000 solution, and Josué after eight days. Several of our animals also showed very early results. No. 10 after five injections of 0.15 c.c. each, No. 32 after six injections of 0.2 c.c. each, and Nos. 24 and 5 after seven and eight injections, respectively, of 0.15 c.c. each.

All these cases and one other (No. 4, in which very pronounced calcification was found) showed inflammatory changes in the lungs and pleura, and some in the pericardium also. Only one rabbit, No. 28, showed similar pulmonary lesions without atheroma, and it differed from the others in the fact that it did not lose weight during the experiment, and seemed in good condition until the end.

As injections of adrenalin are known to cause œdema and punctate hemorrhages in the lungs, we may, with some degree of probability, connect the pulmonary lesions with the injections, and

may draw the following conclusions: Either (*a*) pneumonia and pleurisy favor the development of sclerotic changes in the aorta, or (*b*) those rabbits which are most susceptible to the toxic action on the lungs are most liable to its effects on the aorta.[1]

It also appeared that those rabbits which lost weight showed vascular lesions sooner and to a more marked degree than those which remained in good physical condition. This may be either because systemic disturbance makes the animals more susceptible to the action of adrenalin, or because the latter interferes with the general metabolism of those animals in which its local action is most manifest. Of the fifteen animals which lost weight, ten showed aortic lesions; while of the seventeen which did not, only seven showed such—41 per cent. as compared with 66 per cent.

5. INFLUENCE OF PYROCATECHIN. This substance, dissolved in normal salt solution, was injected intravenously in steadily increasing doses in a series of four rabbits. Only one of them died during the course of the experiment, and as it received less than one-tenth as much as the others, it seems probable that the death was due to some other cause.

The injections were begun with only 0.2 c.c. of a 1:1000 solution, but were slowly increased to 2 c.c. of a 1:100 solution. Doses of 1 c.c. of this solution and upward caused clonic convulsions, and three rabbits received twenty-two such injections each. In spite of the enormous dosage the three rabbits retained their health and weight to the end.

In the rabbit which died early in the experiment (after thirty-one days and sixteen small injections) and in one of the others there were pinhead-size spots which may have been beginning aortic sclerosis, but were probably not. The others showed no sign of any lesion.

We may thus conclude that if pyrocatechin has any deleterious action on the walls of the bloodvessels it is extremely insignificant and not at all to be compared with that of adrenalin.

These results agree with those mentioned in regard to the etiological significance of high blood pressure, and suggest that experiments with a series of bodies closely related to adrenalin, but not causing any increase in blood pressure, might materially advance our knowledge of the mode of its toxic action.

Forty rabbits were used in these experiments, but four of these died too early to be of value, and four were used for pyrocatechin. Of the remaining thirty-two, seventeen showed aortic lesions and ten had calcification; fifteen showed no result. The average duration of the thirty-two experiments was thirty-one days; fourteen lived longer than thirty days, and of these, eleven showed sclerosis;

[1] It is intended to determine later whether lesions of the lung occurring in rabbits not injected with adrenalin are likewise connected with aortic changes.

eighteen died within thirty days, and of these only six showed changes; sixteen received more than 3 c.c., and of these, eleven showed lesions; sixteen received less, and of these, six showed lesions. In the former case 78.6 and 33⅓ per cent.; in the latter 69 and 37.5 per cent. This shows the greater effect of time as compared to amount of adrenalin.

It is perhaps not fair to make direct comparisons between the different series on account of the fact that the ureter ligation rabbits all died within twenty-eight days, and all the thyroidectomy rabbits and pregnant rabbits lived beyond that time. The chromate rabbits lived on the average nineteen days, and the controls thirty-five days. This largely explains the reason that a larger percentage of the thyroidectomy rabbits and controls showed lesions than of the chromate and ureter ligation series.

In generalizing on the whole series of experiments we would like to draw attention first to the fact that although the relation of a number of the factors has been determined there still remains a marked variability in the reaction which we cannot yet explain. The possibility must, of course, be considered that different samples of adrenalin solution varied in toxic power, but even animals injected at the same time with the same solution showed extreme differences. This individual variation may also show itself in the reaction to a single injection. Thus, in one rabbit fatal œdema was caused by the injection of 0.3 c.c., while in other cases as much as 1 c.c. followed by only slight pulmonary œdema.

In one case, after a rabbit had ceased breathing and the heart action was no longer perceptible, rhythmic pressure on the chest wall caused a complete and surprisingly rapid recovery. Whether this was due to artificial respiration or to massage of the heart it is impossible to say.

As positive factors in determining the amount of reaction we can state with some certainty that:

1. Injections given at intervals of four days have more effect than the same number of injections of the same size given closer together.

2. Granted the injection of a certain minimum dose which is able to cause aortic lesions, even a marked increase in the size of each dose causes little, if any, increase in the toxic action. The increase, if any, is markedly out of proportion to the amount injected.

3. Injections given every four days have almost as much effect as injections given every day for the same length of time.

Thus, we see that in determining the result the length of time is the most important factor, the number of injections much less, and the size of the dose still less important. We give these latter conclusions with a certain reserve, as the factor of individual susceptibility complicates the effect of the injections.

Our results agree with those of other observers that the lesions

are limited almost, if not entirely, to the aorta. In most cases patches were found in the arch and descending thoracic. In four cases (Nos. 5, 13, 30, and 32) the abdominal was also involved, and in one of these (No. 13) the change in the thoracic was minimal or doubtful. In No. 27 there was only one small placque, that being in the descending thoracic. In four cases (Nos. 1, 4, 22, and 29) the only lesion was in the ascending portion or arch. In one of these (No. 1) the only spot was in the sinus of the valsalva. In No. 22 there were two small aneurysms.

Microscopic sections were made of aortas in different stages of degeneration, fixed in formalin and stained by hæmatoxylin and eosin, and by Weigart's elastic-tissue stain.

In early cases we find only loss of nuclei in the muscle cells of the media over larger or smaller areas. These cells may be partly vacuolar. In one specimen there were signs of amitotic division of nuclei at one place near the adventitia. In a few sections the internal elastic membrane seemed to be split into a number of fine lamellæ. Whether this has any connection with the other changes and is of any significance only further study can show, as in advanced cases it was still well preserved.

In more advanced cases impregnation with lime salts was found in the central part of the media in the form of larger or smaller plates. The nuclei of the muscle cells had entirely disappeared. The elastic fibres may be well preserved, but they generally loose their wavy appearance and run straight through the calcified area. They may be pushed together as if the substance separating had been lost, and may even look as if glued together. In other cases they begin to disintegrate and may almost disappear. These changes in the elastic fibres may occur in areas near calcareous plates where calcification itself has not yet begun. At some points ruptures had occurred in the incrusted tissue and the elastic fibres with the tissue between was bent into the shape of a shepherd's crook. Changes in the intima and in the adventitia may be absent or very slight even in advanced cases.

Inflammatory processes were never found. There was no round-cell infiltration in any of the coats and no new formation of blood-vessels or connective tissue. This absence of changes of an inflammatory nature is characteristic of the early stages, but collections of certain cells may be found sometimes in later stages around the calcareous plates, as described by Erb. We cannot exclude that new formation of connective tissue may, perhaps, occur in the intima, but it certainly is not constant, as it was not found in our specimens.

We see, therefore, that the character of these lesions differs essentially from the findings in arteriosclerosis and atheroma in man, where the so-called inflammatory changes are very prominent, and where, according to some observers, the initial change is in

the elastic membrane and degenerative processes in the intima are of more importance than those in the media.

Although the difference in histological appearance between atheroma in man and the changes in the aorta of rabbits treated with adrenalin is so great, we cannot positively state how fundamental the differences are or whether they may not after all be of only secondary importance.

ABSTRACT FROM PROTOCOLS.

Series I. Thyroidectomy.

1. Weight, 2260 gr.; at death, 2080 gr. Operation four days before first injection. Duration forty-three days. Adrenalin nineteen injections; 3.9 c.c. Killed. Autopsy showed small diseased areas in the thoracic aorta and one calcareous patch in the sinus of Valsalva.

2. Weight, 2100 gr.; at death, 1760 gr. Operation nineteen days before first injection. Duration fifty-three days. Adrenalin twenty-eight injections; 6.4 c.c. Killed. Autopsy showed extreme calcareous change in the entire descending thoracic aorta. This case shows especially well the fact that thyroidectomy does not prevent the action of the adrenalin, it being, perhaps, our most marked case.

3. Weight, 1960 gr.; at death, 1900 gr. Operation ten days before first injection. Duration fifty-three days. Adrenalin twenty-eight injections; 6.4 c.c. Killed. Autopsy showed no alteration in the aorta. This is especially interesting, as it agreed in all details with No. 2, but showed such a very marked difference in the final result. It shows especially well the danger of trusting to conclusions drawn from a small number of investigations.

4. Weight, 1620 gr.; at death, 1800 gr. Operation seven days before first injection. Chromate begun on twenty-first day. Six injections; 37 c.c. Duration fifty-three days. Adrenalin twenty-eight injections; 6.4 c.c. Died. Autopsy showed pneumonia of lower right lung. Slight disease of ascending thoracic. This case is especially interesting as it bears on several points. In the first place, although it died of pneumonia, it did not show marked lesions; but this fact seems to be counteracted by the remarkable gain in weight. Finally, it shows the lack of effect of the chromate.

Series II. (A) Ureter Ligation.

5. Weight, 1700 gr. Operation four days before first injection. Duration twenty-five days. Adrenalin eight injections; 1.2 c.c. Died. Autopsy showed right-sided pneumonia, bilateral pleurisy, and fibrinopurulent pericarditis. Also an apparently degenerative (fibrous?) patch in the wall of the heart. Aorta diseased throughout. Extreme calcification of the descending thoracic, which affected

somewhat the origins of the intercostals. This case is of extreme interest. It is the only one of this series which showed any lesion in the aorta, but as this was so very extreme and occurred after so small an amount of adrenalin, it emphasizes again the difference in individual reaction. The association of pulmonary lesions is of great interest. The fact that no other of this series showed vascular changes makes it probable that the ligation of the ureter had nothing to do with the extreme alteration. The weight at death was, unfortunately, not taken.

6. Weight, 1400 gr. Operation three days before first injection. Duration forty-three days. Adrenalin twelve injections; 1.8 c.c. Killed. Autopsy negative.

7. Weight, 1600 gr. Operation four days before first injection. Duration nineteen days. Adrenalin five injections; 0.75 c.c. Died. Autopsy negative.

8. Weight, 1800 gr.; at death, 1420 gr. Operation three days before first injection. Duration twenty-five days. Adrenalin seven injections; 1.05 c.c. Died. Autopsy showed doubtful or minimal change in the aorta. In spite of the great loss of weight this rabbit did not show itself susceptible. (See remarks on No. 24.)

Series II. (B) Chromate of Potassium.

9. Weight, 1900 gr. Chromate 1 per cent. Nine injections; 20 c.c. Duration thirteen days. Adrenalin four injections; 0.6 c.c. Died. Autopsy showed no lesions.

10. Weight, 2200 gr. Chromate 1 per cent. Ten injections; 30 c.c. Duration sixteen days. Adrenalin five injections; 0.75 c.c. Died. Autopsy showed double pneumonia and pleurisy. Aorta diseased with calcified plates in the thoracic portion. This is the smallest amount of adrenalin which gave rise to lesions in the course of our experiments and pulmonary lesions were found.

11. Weight, 1400 gr.; at death, 1200 gr. Chromate five injections; 25 c.c. Duration twenty-seven days. Adrenalin ten injections; 1.5 c.c. Killed. Autopsy showed one small calcified spot in the arch and a large diseased patch along the descending thoracic aorta. (See remarks on No. 23.)

12. Weight, 1340 gr. Chromate four injections; 15 c.c. Duration fourteen days. Adrenalin five injections; 0.8 c.c. Died. Autopsy showed no vascular lesions.

13. Weight, 1830 gr.; at death, 1660 gr. Chromate eight injections; 40 c.c. Duration thirty-eight days. Adrenalin fourteen injections; 3.2 c.c. Killed. Aorta showed minimal changes in the thoracic aorta and considerable calcification in the abdominal. This is controlled by No. 17, which showed no pathological changes and which has been pregnant.

14. Weight, 1900 gr. Chromate five injections; 7 c.c. Duration

nine days. Adrenalin four injections; 1.1 c.c. Died. Autopsy showed no lesions of aorta.

15. Weight, 1400 gr. Chromate three injections; 12.5 c.c. Duration ten days. Adrenalin five injections; 1 c.c. Died. Autopsy showed no vascular lesions.

16. Weight, 1740 gr.; at death, 1800 gr. Chromate six injections; 65 c.c. Duration twenty-nine days. Adrenalin twenty injections; 13 c.c. Killed. Autopsy showed no vascular lesions. Interesting on account of the large amounts of adrenalin and chromate and the absence of lesions.

Series III. Pregnancy.

17. Weight, 2060 gr.; at death, 2240 gr. One day pregnant before first injection (?). Duration thirty-two days. Adrenalin eighteen injections; 3.85 c.c. Killed. Showed signs of recent delivery. Autopsy showed no vascular lesions. Injections were started at about the beginning of pregnancy and were given at first every other day, later, every day. She was killed because of a sudden loss of weight, which had been above 2300. The absence of lesions in spite of large amounts of adrenalin is noteworthy.

18. Weight, 2780 gr.; at death, 2720 gr. Two days pregnant before first injection (?). Duration thirty days. Adrenalin seventeen injections; 3.7 c.c. Killed on account of loss of weight, which had risen to 3060. Autopsy showed one small placque in aorta. Remarks as for No. 17.

19. Weight, 2700 gr.; at death, 2550 gr. Five days pregnant before first injection (?). Duration twenty-two days. Adrenalin nineteen injections; 7.8 c.c. Killed on account of loss of weight, but found to be still pregnant and about five days from term. Autopsy showed no calcification; descending aorta appeared uneven. Fetuses normal.

20. Weight, 2000 gr.; at death, 1900 gr. Four days pregnant before first injection (?). Chromate begun early. Seven injections; 20 c.c. Duration twenty-six days. Adrenalin thirteen injections; 3 c.c. Killed after birth of three normal young. Autopsy showed no lesion in mother or young.

21. Weight, 1800 gr.; at death, 1660 gr. Four days pregnant before first injection. Chromate begun early. Six injections; 65 c.c. Duration twenty-nine days. Adrenalin twenty injections; 13 c.c. Killed on account of sudden loss of weight, which had been 1850. Uterus recently pregnant. Autopsy showed no vascular lesions.

The above cases are interesting as showing the protective influence of pregnancy and the negative effect of chromate.

22. Weight, 2000 gr. Eight days pregnant before first injection. Chromate begun only a week before death. Two injections; 10 c.c. Duration sixty-six days. Adrenalin twenty-one injections; 3.9 c.c.

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A STUDY ON BRACHIAL BIRTH PALSY.[1]

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HISTORICAL INTRODUCTION AND CRITICAL DIGEST OF THE LITERATURE OF BRACHIAL BIRTH PALSIES (L. P. CLARK).

Notwithstanding the fact that Duchenne first described brachial birth palsy due to traction in his work on localized electrization in 1872, there is evidence that arm palsies occurred in the process of delivery nearly two hundred years ago. Indeed, a careful review of obstetric works of a still earlier date would probably disclose cases of brachial birth palsy. The palsy is undoubtedly as old as forcible intervention in obstetric practice. Indeed, the association of the two facts early presaged the cause and effect relationship which is known to exist between them in our own time.

A case of compression paralysis of the arm is recorded in an old obstetric report as early as 1768 (Smellie). The labor in which it occurred was a face presentation. Owing to prolonged compression to which the child had been subjected both arms remained paralyzed

[1] Grateful acknowledgment is made to Dr E A Sharp for photographs of the cases, and to M Peterson for the anatomical drawings of the operative field A preliminary report was made before the Neurological Society of New York in April, 1904 A special paper on the pathology was presented before the same society in November, 1904 This paper reports the cases only to January, 1905 Final results will be reported in a future communication

Autopsy showed five or six small patches in the upper part of the aorta.

30. Weight, 1300 gr. Lost weight, but no exact record. Duration thirty-four days. Adrenalin ten injections; 1.5 c.c. Died. Autopsy showed abscesses under the skin of the abdomen. The wall of the left ventricle appeared degenerating. Aorta, about twelve calcareous patches in the thoracic and one in the abdominal. Almost similar to No. 5, which showed about equally severe lesions. Interesting as compared to No. 29, as it was so much more affected by less than half the amount of adrenalin. A case in favor of the view that duration is of more influence than anything else.

31. Weight, 1240 gr.; at death, 1140 gr. Duration twenty-one days. Adrenalin twelve injections; 3.8 c.c. Died. Autopsy showed no vascular lesions.

32. Weight, 1520 gr. Lost weight, but no exact record. Duration ten days. Adrenalin six injections; 1.3 c.c. Died. Autopsy showed disease of the arch and nodule of consolidation in apex of lung, and fibrinous pericarditis. The latter changes explain probably the aortic lesions found ten days after the beginning of the experiment.

Series V. Pyrocatechin.

33. Weight, 1300 gr. Killed. Weighed at death 1500 gr. Duration fifty-seven days. Pyrocatechin thirty-eight injections; 288 c.c. Killed. Autopsy showed no aortic lesions. Gained in weight, notwithstanding the large doses of pyrocatechin.

34. Weight, 1920 gr. Killed. At death, 1740 gr. Duration fifty-seven days. Pyrocatechin thirty-eight injections; 288 c.c. Killed. Autopsy showed no aortic lesions.

35. Weight, gr. 1560 gr.; at death, 1310 gr. Duration thirty-one days. Pyrocatechin sixteen injections; 19.3 c.c. Killed. Aorta showed a few pinpoint-sized opaque spots which were possibly not of the same character as the vascular lesions caused by adrenalin. Animal had lost much weight. Lungs showed scattered areas of solidification.

36. Weight, 1650 gr.; at death, 1625 gr. Duration fifty days. Pyrocatechin thirty-three injections; 258 c.c. Killed. Aorta showed one pinpoint-sized opaque spot which was possibly of the same character as the ones caused by adrenalin.

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The work of some of the authors mentioned below is of such recent date that only references of reports on communications made to societies could be given.

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macher, Kustner, Thorburn, Ballenhagen, Hochstetter, Fieux, and many others. (See appended bibliography.)

Following Duchenne's work, Seeligmüller (1874) presented the next case of laceration brachial birth palsy. Although Erb (1874) reported one case of this type in his original presentation of the adult lesion, the case was not published until after that of Seeligmüller. A fact of greater interest than the simple report of cases at this time was brought up by Fritsch, who in the discussion of Erb's thesis cites a case of arm paralysis in a child two days old, associated with a hæmatoma at the lower portion of the sternomastoid muscle (Erb's point). The case possibly foreshadowed the hemorrhagic nature of the lesion, which until in the present thesis has never been mentioned in the literature of the subject. If so, it must have been a very gross form of the lesion, as the cases here presented gave no history of such palpable birth injury. Moreover, the paralysis in Fritsch's case disappeared with the absorption of the hemorrhagic mass. The accident occurred in a head presentation with forcible forceps delivery. The case is all the more interesting as it was but a single illustration of a series studied by Fritsch in connection with hemorrhagic extravasations in this locality. He had repeatedly seen superficial hæmatoma about the shoulder in breech cases and had inferred that deeper lesions of the same hemorrhagic character should have existed. With the exception of the case mentioned above, his series consisted of stillborn infants; therefore, paralysis could not appear. No histopathological study of the lesion was made. Had this been done the exact nature of the lesion in laceration brachial birth palsy must have long since been demonstrated. The gross appearance of hemorrhage mentioned by Fritsch was not present in either Erb's or Seeligmüller's cases, although the latter was a bilateral lesion of the plexus, occurring after a difficult extraction. No traumatism or complications were associated in the case. Seeligmüller (1877) reported three cases of lower arm type of brachial birth palsy, in which a paralysis complementary to that of Erb was present—*i. e.*, paralysis of the muscles supplied by the lower roots of the plexus. The palsy was associated with ocular symptoms such as narrowing of the pupil and palpebral fissure on the same side as the lesion. This type of brachial palsy was first described in the adult by Flaubert (1827). In Seeligmüller's case the infant was born unaided, but after a very protracted labor. Probably the lesion was purely a pressure one, as the child completely recovered in a few weeks. It is of interest to note that Erb's single case of laceration brachial birth palsy reported in 1874 and published in 1877 was thought to be due to pressure of the hooked finger in the axilla, as practised in the Prague method of version and forcible extraction. The inconsistency of a permanent palsy (over two years old at time of report) being induced in the fifth and sixth nerve root by pressure in the axilla was apparently not appre-

for several days. Discussions and theses at an early date bear testimony of the needs of classification of the causes and the resultant types of obstetric birth palsies. Thus some cases which apparently showed a wide range and severe degree of palsy at birth recovered in a few days, where others of slighter apparent defect recovered but little or not at all after years of medical treatment. A significant case of the former type is that of Jacquemier (1846), in which the palsy of the upper arm recovered in twenty days, while the first case of Duchenne-Erb palsy reported by Danyon in 1851 illustrates the severe destructive nature of the typical lesion. In this case, after an apparent simple compression which induced the palsy, the child died on the eighth day. A profound and extensive hemorrhagic infiltration was found in the entire brachial plexus. The condition found at autopsy was strongly suggestive of a lacerating lesion of the brachial plexus. Still other cases antedating Duchenne's report are those of Doherty (1844), Mattei (1862), Guenoit (1864), and Depaul (1864). The case by the two latter authors was reported in 1864 by each separately, and, owing to error of subsequent writers, has done double duty, being counted as two cases. Although compression was alleged as the cause, the unfavorable outcome leads one to suspect that it too, as in Danyon's case, was the result of a stretching and lacerating of the plexus. While Duchenne recognized three forms of brachial palsy in the newly born, he expressly outlines the clinical syndrome of the type classed in this study as "laceration brachial birth palsy."

(In this study the cases are classified as (1) "compression birth palsy," the result simply of pressure; and (2) "laceration birth palsy," from overstretching of the nerves with resulting laceration in varying degrees of the nerve sheaths and their contained vessels as well as of the nerve fibres themselves.)

Duchenne saw a few cases of compression palsy and relatively few of a subspinous dislocation of the humerus. As Duchenne had no definite conception of the nature of the nerve lesion present in any of these palsies, a more exact determination of the relationship of the different causes and effects was not possible at that time. He had no doubt, however, that in his five cases of obstetric palsy traction was the sole factor in its production, inasmuch as no forceps were used in the delivery and great pulling of the head by hand was employed in the process of extraction. Two of his cases were breech presentations. Duchenne saw all of the five cases in consultation with the obstetricians in whose practices these nerve accidents occurred.

Immediately following Duchenne's work upon these conditions, four cases were reported by Nadaud, including a case each of Bailly's, Tarnier's, Chantreuil's, with one of his own.

After Erb and others had described this same type of paralysis in adults, cases of obstetric palsy were reported by Schultz, Schu-

styled the disease under a new name, "spinal paralysis," and regarded it as due to a "rare type of anterior poliomyelitis." The discussion failed to correct the diagnostic error. Ten cases were reported under this head, which were all typical of laceration brachial birth palsy. A paper of extreme interest is that of Guillemot (1897), in which he reports a veritable epidemic of obstetric birth palsies in a particular locality covering a term of years. There were no less than 30 cases in all delivered by the same midwife by podalic version. Guillemot saw 12 of these cases, most of whom suffered from the permanent or laceration type of palsy. They were all adolescent or adult cases. Two of the cases were bilateral in type. In his research 2 cases in each of two families were noted. In those days no particular explanation seems to have been expected from the obstetrician to account for any of these accidents. A sufficient number of cases had been reported by 1897 for Cibert to collect a series of 66, but nothing new was brought out. Studies upon the different types of obstetric palsy soon began to appear. Seeligmuller and Thorburn reported the "Klumpke paralysis" (1885), which should properly be called after Flaubert, who first described the condition in 1827. The type consists of a lower brachial plexus lesion. In 1896 Weil published a valuable article upon the differentiation of superior arm, lower arm, and mixed-arm types of birth palsies and their various combinations in the same case. He gives a very clear account for the first time of an entire cervicobrachial lesion.

In relation to the possible extent of the lesion, aside from that in the plexus nerves, the work of Barbarosa di Grovellona may be mentioned here. The possibility of there being a cord lesion associated as the result of the heavy traction employed in delivery is worthy of attention. Barbarosa places on record an entirely new form of obstetric palsy. The brachial paralysis he reports is accompanied by cord lesions. The cases illustrating this type consisted of double brachial monoplegia and amyotrophy as a result of the cord lesion (?). The condition was not common. It would seem that an error of diagnosis had been made. He inferred that the violent stretching in laceration birth palsy may exceptionally produce a tearing of nerve roots at or in the cord itself. The possibility of this occurrence will be dealt with in the experimental work on etiology in this research.

Inaccurate as the early statistics must have been it is of interest to note that of the 95 cases collected by Schümacher (1899), 53 were head and 40 breech cases; of the 53 head cases, forceps were used in 28; spontaneous births, 10; history of finger hooked in axilla, 10; history of traction with head laterally inclined, 8. Of the 40 breech cases 6 were of spontaneous delivery and 34 assisted.

In a careful review of the literature it is evident that with very few exceptions obstetric palsy is due to manipulation in delivery.

ciated by Erb, notwithstanding the fact that his thesis was specially directed toward proving that his type of adult palsy was caused by none other than pressure of the clavicle upon the fifth and sixth cervical roots in the neck.

Nothing essentially new is contained in the report of cases of Bernhardt (1880) and Thorburn (1886). The first monographic consideration given to the subject was that of Hamond (1881); but the work sheds no new light upon the nature of the lesion. The same may be said of Roullaud's (1887), although a number of new cases were reported. The elaborate work of Kustner (1889) still repeated the dictum that the persistent Duchenne's obstetric palsy must be secondary to bone injuries, especially fracture at the upper epiphysis of the humerus. The absence of all subsequent evidence of a birth fracture in these many cases did not seem to have rendered the author any the less certain of the etiology of the palsy. He believed, however, that the cases which recovered spontaneously were due to forceps compression. No mention is made of stretching or rupture of the plexus. This reactionary view appeared to suppress research work for some time, as but little was done on the subject in the next few years. Schultz published the first article directed specifically to pathogenesis. Erb's theory was generally accepted at the time. Schultz reported a case of arm paralysis in a child seen when two years old, with a history of breech extraction and bringing down of the right arm behind the head. The case was typical of the laceration palsy herein described. Schultz applied Erb's theory of clavicle compression and elaborated a defence of this view. The inaugural dissertation of Arens (1889) at Göttingen upon "Obstetric Paralysis" is worthy of passing mention, as it was a monographic treatment of the subject at the hands of an American. But one new case was reported. It was a breech presentation delivered by the Veit-Smellie method. A bilateral Duchenne-Erb palsy resulted. The left side recovered rapidly. Arens assumed the presence of a deep-lying hæmatoma on the left side which, as recovery took place, underwent absorption. He conceived that the right plexus had sustained a more severe injury (rupture?). There appears to be a gap in the literature between 1889 and 1897, the date of Fieux's essay upon the etiopathology of obstetric palsies. The experimental work of this author leads him to depart strongly from Erb's theory of compression. He believes that overstretching is the chief causal factor. (The experimental work on the etiology described later was done and the conclusions formulated before the thesis of Fieux had been discovered in the literature.)

During this quiescent period (1889-1897) Comby and Jaffroy (1891) reported cases, as did also several American authors (Lovett and Burr, 1892). Burr's report ignored all the work previously done, as well as that of Lovett, which was published in the same volume of *Transactions of the Boston Obstetrical Society*. Burr

nerve roots. Also the pectoralis major and deltoid muscles, with skin, fat, and fascia were removed below the clavicle. This gave a free exposure of the brachial plexus, vessels, and bony structures.

The following anatomical facts were noted:

(*a*) The clavicle is small, has a smooth, rounded, posterior surface, is so curved as to fit around the side of the neck without localized pressure, is somewhat flexible, and the ligaments of its articulations at both ends are so loose as to allow free play of the bone in any direction.

(*b*) The first rib is small, flat, and very flexible.

(*c*) The nerve roots come out between the transverse processes of the cervical vertebræ, of which the anterior tubercles lie well forward of the nerve roots.

(*d*) The nerve roots lie between the scalenus anticus in front and scalenus medius muscle behind, which form a protecting muscle cushion.

(*e*) There is a thick layer of fat and fascia behind and below the clavicle.

(*f*) The clavicle lies well below the usual site of lesion in brachial birth palsy (*i. e.*, fifth and sixth roots) and can be pushed up to that site only with some difficulty.

(*g*) When the clavicle is pushed directly backward with the arm at the side, or when it presses backward during hyperextension of the arm, (1) the nerves slide outward away from the lines of pressure; (2) the nerves are pressed upon so little that they slide up and down behind the clavicle with the greatest freedom when traction is applied to them.

(*h*) In hyperextension of the arm the clavicle rotates backward and slightly downward. It does not rise to the level of the lesion commonly found in brachial birth palsy. The nerves are not rendered tense by this manipulation.

Reasoning from these data, backward pressure by the clavicle is not a factor, inasmuch as the bone does not reach to the level of the usual lesion; the anterior tubercles of the transverse processes prevent it from pressing the roots at their exit from the spine; the first rib is so flexible as to give scarcely any resistance to pressure; the nerves are surrounded and protected by muscle and fat cushions, and the clavicle is so curved, so smooth posteriorly, and so loosely attached at either end that when it is displaced strongly backward the nerves can still be easily moved either sideways or up and down, showing an absence both of pressure and tension.

If, however, in exceptional cases, the nerve should be compressed by the clavicle, the result would be a narrow transverse lesion of moderate degree which would always go on to spontaneous regeneration. Moreover, if the lesion were the result of compression between the clavicle and first rib, the lower roots of the plexus should show most damage because they are so placed as to sustain the

Obstetric palsies form a class entirely by themselves and have nothing to do with cerebral or other congenital palsies. As a rule, they occur in superior arm most frequently. More rarely the lower-arm type has occurred; very rarely total palsy is reported (10 cases). Now and then a case of individual muscle paralysis has been reported in obstetric palsy.

ETIOLOGY (A. S. TAYLOR).

The predisposing factors in the production of this lesion are such disproportion between the size of the child and the maternal pelvis as would delay the easy progress of the labor, and especially as would hinder the rotation and descent of the shoulders after descent and extrusion of the head, rigidity of the maternal soft parts, maternal exhaustion, and any other cause which will delay labor and therefore lower the vitality of the fetal tissues.

Apparently, from the cases on record, transverse and face presentations have not been responsible for the production of many of these cases.

They occur with about equal frequency in vertex and breech presentations. (Schümacher, 93 cases: 50 vertex, 43 breech.)

Brachial birth palsy occurs in about one in two thousand labors (Duvall and Gullian, 1901). If one admits that laceration birth palsy occurs in but one-half of all the cases, the ratio stands one in four thousand for that type of palsy which is the one under consideration here.

It is erroneous to suppose that laceration birth palsy occurs only in mismanaged labor; it may easily result in highly skilled hands, as shown by its occurrence at the birth of Emperor William of Germany, on which occasion Sir William Jenner was the accoucheur.

It is manifestly important to determine the usual site, extent, and nature of the lesion, as well as the etiological factors concerned as exciting causes of the lesion.

Those factors commonly mentioned are:

I. Backward pressure on the nerves by the clavicle, (*a*) on transverse processes of the vertebræ; (*b*) on first rib.

II. Hyperextension of the arms in breech cases.

III. Pressure of forceps.

IV. Tension on the nerve roots.

It is noticeable that, while so many different causative factors are cited, the results are invariably the same in type, though differing in degree. This strongly suggests that one fundamental type of nerve injury underlies all the cases.

To determine which of these factors is the essential one, twenty dissections were made upon ten infants within three to ten days after death. The skin, fasciæ, and fat were removed from the lower part of the posterior cervical triangle to fully expose the clavicle and

very few cases are known where, after a normal vertex labor with neither instrumental nor manual traction, a temporary birth palsy has occurred.

In breech presentations the lesion occurs in the delivery of the after-coming head. The fingers hooked over the back of the neck pull both shoulders down and away from the head and neck. Here again rotation or oscillation increases the strain on the nerve roots. Moreover, the tips of the fingers, hooked over the shoulders, lie upon the stretched nerves and add a lateral strain to the tension they already suffer from.

It is evident then that any form of traction which pulls the head and neck away from the shoulder is the exciting cause of the lesion. As a natural corollary the lesion may occur with any "presentation."

It was most interesting to note the nerve area involved and the sequence of events in the production of the lesion on the cadaver.

It was invariably the case that the fifth root gave way first, then the sixth, and so on down the plexus in regular order if the force used was sufficient. From the lay of the plexus it is perfectly obvious that the fifth root must bear most of the strain until it yields, then the sixth, and so on downward.

This same fact is borne out clinically, inasmuch as the mildest cases show only paralysis of muscles belonging to the fifth root and progress from that to cases involving rupture of the entire plexus.

The nerves do not give way in a limited transverse section, but fray out over a considerable linear area, just as does a rope when it is overstrained. This fact accounts perfectly for the pathological pictures found in our seven cases at the time of operation.

The location of the lesion is important and will be more easily understood if it is remembered that the fifth and sixth roots fuse about 1.5 to 2 cm. (in infants) from their spinal exits. Their junction is from 0.5 to 1 cm. long, and from its distal end pass two nerves to form part of the outer and posterior cords of the plexus respectively. For convenience sake, these may still be called the fifth and sixth nerves, and in locating the lesion the terms "above," "at," and "below the junction" will be used.

In the twenty experiments cited the lesion was produced:

	Fifth nerve.	Sixth nerve.
Above junction	16 = 80 per cent.	17 = 85 per cent.
At junction	2 = 10 "	2 = 10 "
Below junction	2 = 10 "	1 = 5 "

In the case of the sixth nerve, 7 (35 per cent.) of the ruptures consisted in avulsion of its root, including the root ganglion from the spinal cord.

Great force must be employed to cause the lesion, which accounts for the relative infrequency of its occurrence.

Of the 7 operative cases, 1 (Fig. 6, Case I.) involved rupture of the entire plexus. Of the remaining 6, 5 (83 per cent.) showed the

maximum damage above the junction and 1 (17 per cent.) below it, about the same percentages as in the experimental cases. In all the cases the cicatricial sequelæ of the injury involved the "junction."

In 1 case (Fig. 6, Case VII.) the fifth root was torn across above the junction, the ends separated about 1 cm., and bound down by connective tissue.

In 1 case (Fig. 8, Case III.) the fifth root was torn across just below the junction, and the distal end displaced inward and downward about 2.5 cm. to the front of the scalenus anticus muscle, where it was adherent.

There was no case of avulsion of the sixth root.

It will be seen in these cases that the maximum damage is to the fifth root.

A nerve of special importance which is always involved is the suprascapular. It is a small nerve arising from the distal and outer aspect of the junction of the fifth and sixth roots and supplies the external rotator muscles of the shoulder.

PATHOLOGY (T. P. PROUT).

To properly appreciate the lesion in these cases the etiological importance of the overstretching process in its production at birth must be borne in mind. Furthermore, a study of the lesion should demonstrate not only the characteristics of the degenerated areas, but the reasons for the failure of complete regeneration following the accident. The severed ends are in apposition in the vast majority of instances; when such condition is brought about surgically, union, regeneration, and recovery of function occur without incident. Why are these cases exceptional in this particular?

In this connection several points must be borne in mind:

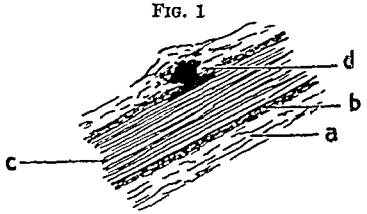
1. The force producing the lesion is variable; the lesion therefore varies in extent with the variation in the extent and character of the force producing it, as has been pointed out. In the milder (most usual) cases only the fifth and sixth cervical roots suffer (Case VI.), while the severer cases present a literal severing of the roots of the plexus, varying in number from one (Case VII.) to three or even more (Case I.).

2. The nature of the force producing the lesion is such that the nerve roots are *pulled* apart rather than severed clean at a given point. The lesion, therefore, is incomplete at any given cross-section of the nerve bundle and involves different fibres at different levels.

3. The nerve bundle is surrounded by a dense sheath of connective tissue, the perineurium, which supports it and the vessels supplying it. In any lesion of the nerve roots resulting from an overstretching process this supporting sheath must first give way. In these cases it is torn asunder and the arterioles belonging to it and supported by it are ruptured. A hemorrhage into and beneath the

perineural sheath and infiltrating the strands of nerve fibres and the meshes of the epineurium results. In the recent state a small hæmatoma infiltrating these structures forms at the point of rupture, as shown in the accompanying schematic diagram (Fig. 1).

FIG. 1



Schematic representation of a rupture of the perineural sheath and hemorrhage into its substance and into the meshes of the epineurium resulting in the formation of a hæmatoma: *a*, epineurium, *b*, perineurium; *c*, nerve bundles; *d*, hemorrhage.

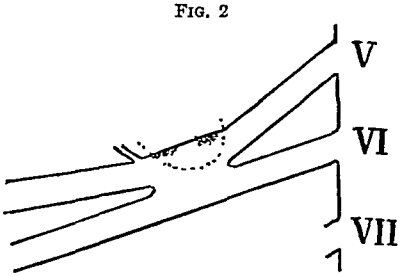
4. This accident occurs at birth, a period of life when the vascular and nervous structures are susceptible of injury of a far-reaching character.

Among these various points only those of immediate import in the production of the lesion interest us here.

Of the immediate lesion little has been written. Strange to say, it is scarcely mentioned in medical literature, there being no adequate description of the gross lesion, and none whatever of its microscopic appearances. The gross appearances vary much with the duration and severity of the case.

In all of the operative cases here detailed the deep cervical fascia was invariably found greatly thickened, especially over the plexus, and irregularly adherent to the nerve roots. The thickening of the fascia was of an irregular and variable character, depending upon the severity of the case.

FIG. 2

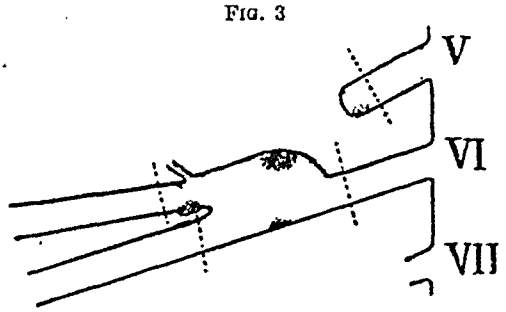


Schematic representation of the position of the gross lesion in Case VI. The dotted line represents the area excised, and the dark areas within it the lesions in the perineural sheath.

In cases of the milder grade, such as Case VI. for example, the outward appearance of the nerve strand at the point of the lesion may be normal, but in this particular instance a distinct nodular mass could be felt in the upper and outer aspect of the nerve strand (Fig. 2).

In this instance also the duration of the lesion was such that the *gross* abnormality was reduced to a minimum. In Case VII., on the other hand, the lesion had existed but eleven months and was, therefore, relatively fresh, and may be taken as presenting some of the more characteristic points of a relatively recent lesion of the severer grade. In this instance there was a severance of the fifth cervical root just before its junction with the sixth root (Fig. 3).

FIG. 3



Schematic representation of the gross lesion and its extent in Case VII. The dotted lines show the area excised. The actual size and shape of the larger piece is shown in Fig. 4. The dark spots represent the positions occupied by the various hæmatomata resulting from the rupture of the perineural sheath and hemorrhage into its substance and the meshes of the epineurium.

In this case the severed ends were more or less rounded and embedded in a hæmatomatous mass, as shown in Fig. 4, which is a drawing of a section through the entire length of the larger piece excised in this case. The larger of these hæmatomata is represented at *a* in the figure, while there are smaller ones at *b* and *c*, all of which represented nodular masses in the gross specimen quite distinct in each instance.

FIG. 4



Actual size of piece excised in Case VII. *a*, small hæmatoma in the perineural sheath and epineurium; *b*, smaller hemorrhage with marked infiltration beneath the perineural sheath; *c*, strands of nerve fibres.

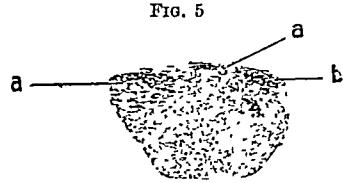
The recognition of the importance of the rupture of the perineural sheath and the resulting hemorrhage into its substance and the substance of the nerve bundle is not mentioned in works upon this subject.

These are points of great importance, since they are *the* determining factors in the production of the ultimate lesion. Were it not for these, we might reasonably expect a recovery more or less complete in all cases except those few presenting an actual rupture of the roots of the plexus. These factors, however, determine the ultimate extent and final character of the lesion.

The extent of the lesion varies greatly with the severity of the

case. Fig. 2 presents in schematic form the extent of the lesion in one of the milder cases. In this instance the lesion occurred at the customary site at the junction of the fifth and sixth cervical roots. The dark areas in the figure represent the extent of the lesion in the nerve sheath as developed in a series of sections lengthwise through the piece excised. A drawing of the entire section slightly magnified, showing the distribution of the lesion, follows in Fig. 5.

FIG. 5



Longitudinal section of piece excised from Case VI., magnified 2: *a*, perineural sheath; *b*, pigment deposits and connective tissue infiltrating the substance of the nerve bundle.

In Case VII. there was a severance of the fifth root. The extent of the lesion and the area excised is shown in Fig. 3. In this instance the lesion was comparatively recent and fairly well organized hæmatomata were present in the nerve sheath, as already mentioned. Fig. 4 shows the larger of the excised pieces, actual size. The position of the various hæmatomata is also indicated. In Case III. the lesion was still more decided, the fifth root being severed below the junction with the sixth and the excised mass (junction of the fifth and sixth roots) being involved in a ruin more or less complete. In this instance the cells of the neurilemma which represent the remains of the nerve fibres are thrown into folds, one of which is shown at *A* in Fig. 12, 3. The fibres *A* come to an abrupt ending in a connective-tissue mass, *C*, an old organized hemorrhage, with some masses of pigment still remaining in the interior.

The types of lesions depicted in these three cases represent fairly well the varying grades usually met with.

A conception of the ultimate lesion is to be obtained only by a study of the sequence of events in the nerve trunk following the primary lesion described and depicted above.

Organization of the blood clot or hæmatomatous mass and a union of the severed perineural sheath take place before any attempt at regeneration of the nerve fibre itself. The new connective tissue formed in this process contracts upon the nerve bundles and prevents regeneration of the nerve fibres. If the lesion in the perineural sheath is considerable, resulting in the formation of hæmatomata at the points of rupture, as in Case VII., the effect of the organization of the blood-clot will be such as to prevent the regeneration of a relatively large number of nerve fibres in the nerve bundle. Such points of compression are illustrated in Fig. 11, Cases II. and V. In the latter instance the lesion was too recent to show the disfiguring effect of its contraction upon the nerve fibres.

In all of the cases thus far examined the evidences of an old hemorrhage still persist in the tissues (Fig. 12, 1, *B*, and 4, *A*). In some of these areas the section presented a fairly large vessel in the immediate neighborhood of the old hemorrhage which may easily have figured in the production of the original lesion.

A further possibility following the rupture of the perineural sheath is shown in Fig. 12, 2 and 4. In the first a dense band of connective tissue, *A*, crosses the nerve bundle and strangles it. In the latter there has been a buckling inward of the perineural sheath as shown at *A*, and also in Fig. 13, 2, *A*. This buckle in the sheath effectually strangulates the nerve bundle and has prevented its regeneration.

As regards the behavior of the nerve fibre caught in lesions of this character, its appearance varies very much with the extent and duration of the lesion at a given point. Fig. 13, 1 and 3, present areas of extreme compression. In the unstained areas nothing remains except the cells of the neurilemma, together with some old fragments of the myelin sheath. 3 presents in particular the results of an incomplete compression of the nerve trunk.

From a study of these sections it is evident that the lesion in these cases is not confined to any one given level, but even in the milder grades of palsy extends over quite a wide area.

Some special features in connection with particular cases deserve notice:

In Case VI. there were two lesions in the perineural sheath separated by a short distance (shown in text, Fig. 5) and varying greatly in consistency. The mass at *b* was a hard nodule quite readily felt in the gross specimen; while the lesion at b^1 was more diffuse and the tissue of looser arrangement. Both of these areas contain some pigment deposits, the remains of an old hemorrhage which originally involved the nerve trunk diffusely and has since undergone organization, choking the involved nerve fibres in a contracting connective-tissue mass.

Case VII. belonged to the severer type and differed from the other cases in which the tissue has thus far been examined in being of comparatively recent origin. In the area excised there were two hæmatomata, each about the size of a small pea, which had developed in the substance of the epineurium (Fig. 4, *a*).

The sequence of events in the production of the laceration brachial birth palsies may be summarized as follows:

The lesions produced are (*a*) immediate and (*b*) remote.

The immediate lesion consists in the tearing of the perineural sheath surrounding and supporting the nerve trunk and the incidental rupture of the bloodvessels belonging to it. Hemorrhage occurs into and beneath the perineural sheath, and in many instances into the substance of the epineurium. There is, furthermore, a severance of the nerve strands more or less complete, depending on the severity of the case.

The remote lesion is brought about and its extent determined by (*a*) the healing of the rent in the perineural sheath; (*b*) the size of the blood clot and its ultimate organization; (*c*) the ultimate contraction of the cicatrix upon the nerve strand, which not only prevents its regeneration, but determines a neuritis in certain of those, not severed, upon which it may chance to impinge.[1]

SYMPTOMATOLOGY AND DIAGNOSIS (L. P. CLARK).

The "laceration palsy" dealt with here is so characteristic in type that in severe cases its clinical recognition, from the attitude alone, is easy; thus the arm hangs powerless by the side, and cannot be abducted at the shoulder because of the palsy of the deltoid and supraspinatus muscles; the forearm is extended, and cannot be flexed on account of paralysis of the biceps, brachialis anticus, and supinator longus; the hand is in extreme pronation, caused by palsy of the supinator brevis and biceps, and the entire arm is so rotated inward that the palm of the hand may look backward and outward. The humerus is markedly rotated inward as a result of paralysis of the supraspinatus and infraspinatus and teres minor muscles.

Where laceration of the nerve roots has occurred there is present a factor of very great clinical importance not heretofore recognized and of fundamental significance in the early treatment and prognosis of the condition. When an infant remains peevish and fretful for a considerable period after this accident at birth, and when handling of the extremity greatly increases the pain and irritability, there is present a traumatic neuritis aggravated by pressure incident to the organization of the blood clot and repair of the rent in the perineural sheath.

Cases that do not present this symptom will show a more or less complete spontaneous recovery; while in those cases presenting these signs of a neuritis a considerable palsy will follow, depending on the severity of the lesion.

In severe cases after several months the internal rotators, especially the pectoralis major, become markedly contractured, further increasing the deformity of the arm at the shoulder-joint, and this, combined with the paralysis of the external rotators, may lead to more or less posterior displacement of the head of the humerus (as in Case III.). There are present also some contracture at the elbow preventing full extension and more or less flexion contracture and ulnar adduction at the wrist. These attitudes are well seen in all the case pictures. The limits of motion are well shown in Figs. 7 and 8.

[1] In some cases of extreme laceration brachial birth palsy, both congenital torticollis and spasmodic wryneck are also present. From the nature of laceration birth palsy and its pathogeny it is probable that the lesion in the above conditions is a nerve injury resulting from compression or stretching, and not a lesion of the muscles as Stromeyer contends. The spasmodic character of these affections can be explained on the basis of incomplete nerve regeneration, as is true of the spasmodic conditions in chronic facial palsy.

The clinical picture of laceration brachial birth palsy (chronic) is well shown in Figs. 7 and 8, 1. The muscles affected do not belong to the distribution of any single nerve trunk of the arm involved. Erb indicated the exact point at which the lesion could be demonstrated, and this point has been called by his name. The site is about 2 cm. outside the sternomastoid and the same distance above the clavicle, as shown in Fig. 8, 4. Erb demonstrated that by means of electric stimulation at this point one may evoke contraction in all the muscles found affected in this type of paralysis. This point corresponds to the junction of the anterior divisions of the fifth and sixth cervical nerve roots. It has been shown by Erb experimentally, and later by others through clinical study, that the motor fibres to the deltoid, biceps, brachialis anticus, supinator longus, and sometimes supinator brevis muscles run in the junction of the anterior divisions of the fifth and sixth nerve. The palsy resulting from an injury of the nerves at this point is obvious. Orientation of the site of the lesion may be facilitated by referring to the semidiagrammatic scheme of the cervical nerve roots and the formation of the brachial plexus by their union (Fig. 9).

In laceration birth palsy the fifth and sixth cervical nerve roots suffer first. Frequently the seventh and occasionally the eighth cervical and first dorsal nerve roots are involved, thus constituting a lesion of the entire plexus as shown clinically in Fig. 6, 1. In not a few cases of the laceration palsy the entire arm is paralyzed, but for a time only. In such, the milder degree of lesion in the lower cervical nerve roots is recovered from in the course of a few months. Sufficient data are not yet at hand to declare the exact nature of the stretching lesion in these lower cervical nerve roots which recover in part. It seems probable, however, that temporary palsy results here in consequence of simple stretching of the nerve strands without causing rupture of the bloodvessels or the perineural sheath itself. Certainly a lesion of the severe type hereafter to be described could not be present in any marked degree and spontaneous recovery take place.

The sensory changes in brachial birth palsy are very slight and do not aid in the diagnosis. The overlapping of the segmental cord supply to the skin in the affected parts may account for the absence of sensory changes. Although careful tests were made, no detectable sensory changes were present in this series of cases either before or after operation.

The amount of atrophy is moderate, but the developmental defect which is noticeable after a time may be slight or severe, and apparently it is more dependent upon the extent of the brachial lesion than upon any degree of local damage to the plexus nerve. A case now under observation shows extreme dwarfing of the entire upper limb, although all the muscle that is now present seems of good quality, and there is but slight limitation of the motions of the ex-

tremity. The diagnosis is based on the history of traction used at delivery, followed immediately by paralysis of the upper extremity, with the assumption of the attitude and characteristics mentioned just previously in the symptomatology. The degree and extent of lesion in any birth palsy case cannot be determined immediately after its occurrence. There is no definite test to determine whether the nerves have been divided, simply compressed, or lacerated and contused by overstretching. Electric tests are only possible in the palsied child when it is placed under anæsthesia, and even then the results of such tests are contradictory and misleading, a fact early recognized (Stransky, Soltman, Westphal). The diagnosis of the range of nerve injury may be made approximately by analysis of the resulting muscle palsy, the atrophy and extent of growth defect. The electric reactions of the muscle (R. D. of paralyzed muscle) also assist in this particular. In the course of a few months one may detect by palpation a thickening and induration of the nerve roots at Erb's point. By palpation a fairly accurate knowledge of the state of the whole plexus may be obtained in those children who are not fat and who assist the examiner by moderately shrugging the shoulders. By practice one soon gains expertness in this diagnostic aid. The following table embraces the most recent data of the neuromuscular anatomy of the parts affected in birth palsy, and aids much in determining the distribution and degree of lesion present in a given case. It should be used in connection with Fig. 9.

This table shows the immediate nerve supply, plexus division, and nerve-root origin of the muscles of the shoulder, arm, forearm, and hand.

MUSCLES MOVING SCAPULA AND ARM.

Muscle.	Immediate nerve supply. *Plexus Division.*	Root origin.
Trapezius.	Spinal accessory.	C. II. to VI.
Rhomboideus.	Spinal accessory.	C. IV. and V.
Levator angulæ scapulæ.	Spinal accessory.	C. III., IV., V.
Serratus magnus.	Posterior thoracic.	C. V., VI.
Deltoid.	Circumflex (posterior cord).	C. IV., V.
Supraspinati and infraspinati.	Suprascapular.	C. IV., V.
Teres minor.	Circumflex.	C. V.
Subscapularis.	Short subscapular.	C. V., VI.
Latissimus dorsi.	Long subscapular (posterior cord).	C. VII.
Pectoralis major.	Anterior thoracics (outer and inner cords).	C. V. and VI.
Teres major.	Suprascapular (posterior cord).	C. VI., VII.

MUSCLES MOVING FOREARM.

Triceps.	Musculospiral (posterior cord).	C. VI, VII.
Brachialis anticus.	Musculospiral and musculocutaneous (outer and posterior cord).	C. V., VI., VII.
Biceps.	Musculocutaneous (outer and posterior cord).	C. V., VI., VII.
Supinator longus.	Musculospiral (posterior cord).	C. V., VI., VII.
Supinator brevis.	Musculospiral (posterior cord).	C. V.
Pronators (teres and quad.).	Median (outer and inner cord).	C. VI., VII.

MUSCLES MOVING THE HAND.

	Flexors of Wrist.	
Flexor carpi uln.	Ulnar (inner cord).	C. VIII., D. I.
Flexor carpi rad.	Median (inner and outer cord).	C. VII., VIII.
	Extensors of Wrist.	
Extensor carpi rad. longior. Extensor carpi rad. brevior. Extensor carpi ulnaris	Musculospiral.	C. VI., VII.
	Extensors of Fingers.	
Extensor communis digitorum.	Musculospiral.	C. VI., VII.
Extensor indicis.	Musculospiral.	C. VI., VII.
Extensor minimi digiti.	Musculospiral.	C. VI., VII.
	Flexors of Fingers.	
Flexor sublimis.	Median (inner and outer cord).	C. VII., VIII.
Flexor profundus.	Median and ulnar (inner, outer, and posterior cord).	C. VII., VIII. D. I.
Interossei and lumbricales.	Ulnar (inner cord).	C. VIII., D. I.
Two outer lumbricales.	Median (inner and outer cord).	C. VIII., D. I.

PROGNOSIS (L. P. CLARK).

The prognosis in birth palsy depends upon the (*a*) nature and degree of the nerve injury; (*b*) the treatment.

(*a*) In the mild cases where possibly compression alone (rare) or at least only the slightest grade of laceration occurs, spontaneous and fairly prompt recovery may be expected (three to nine months). However, even in these cases there may be a persistent atrophy and retarded development through life of a part or the whole of the extremity, although the range of motions may be little or not at all restricted.

In more decided lacerations of the nerves the outcome depends on how much the cicatricial contractions in and about the nerves (see Pathology) obstruct the anatomical and functional continuity of their fibres. There may, then, result any condition from nearly complete recovery to the total loss of function of a part or the whole of the extremity.

Inasmuch as it is impossible, just after the injury (diagnosis), to determine the exact distribution and degree of the lesion, only time (six to twelve months) will permit a fairly accurate prognosis to be made.

In those cases presenting definite "traumatic neuritis" of the nerve roots (symptomatology) the prognosis is relatively bad from the outset, and it may be positively stated that there will be some degree of permanent damage to the extremity, although here again only time can determine its exact extent. In many cases the lesion consists of several foci scattered through the nerves at the site of injury. Therefore the prognosis is influenced by the distribution as well as the degree of the lesion.

The deep cervical fascia overlying the nerve roots is injured in all the severer cases, and by its cicatrization and adhesions to the

nerve roots (noted in all 7 operated cases) interferes with their regeneration.

(*b*) Proper treatment greatly influences the prognosis even in those cases which will suffer from permanent nerve damage, for, as stated in the symptomatology, there is always some recovery in the nerves which have been only moderately damaged about the periphery of the maximum laceration. During the recovery of these nerves and muscles, unless proper treatment (see Treatment later) is carried out, there occur contractures of muscles and ligaments, with resulting deformities of the joints which very greatly limit the functional value of such muscles as regain power.

Only 26 per cent. of all forms of brachial birth palsy recover (Bruns).

It has been stated (Comby) that cases which occur in head presentation have much the better chance of recovery. While this statement is borne out by statistics, yet one can understand that as less stretching force is apt to be applied in vertex labor, these cases would naturally show less serious lesions

TREATMENT (CLARK AND TAYLOR).

(*a*) *Prophylactic.* The necessity of prophylaxis is obvious and it can easily be deduced from a consideration of the "etiology."

(*b*) *Palliative.* Inasmuch as the degree of spontaneous recovery which will occur in any given case cannot be determined until after many months, treatment should be directed to maintaining the nutrition of the muscles, to preventing contractures and resulting deformities, and to assisting the spontaneous repair of the damaged nerves by means of massage, passive motion, douches (hot and cold), electricity, and such apparatus (if necessary) as will overcome a tendency to deformity.

It must be remembered, however, that in the cases showing marked evidences of traumatic neuritis, complete immobilization is demanded until the inflammatory reaction has subsided (often two to four months). During this period means should be taken in the immobilization to hold the extremity in normal position (*i. e.*, overcome the internal rotation) and so prevent contracture of the pectoralis major muscle and the anterior ligaments in that attitude. The attempt to use the active measures above detailed tortures the child and aggravates the inflammatory reaction. When the neuritis has subsided the active measures may be employed.

Electricity not only helps keep the muscles in good condition, but also aids materially in the nerve regeneration by stimulating "neurofibrillation" in the regenerated homogeneous axis cylinders (Bethe).

(*c*) *Radical.* The length of time through which palliative treatment should be continued is still a matter for discussion. If, by the

end of the year, complete recovery has not occurred, a prognosis of permanent lesion may be definitely made, although some slight improvement occurs after this time in certain cases.

Kennedy counsels operation at the end of three months, but that would seem too early, inasmuch as, at that time, none of the spontaneous regeneration, which occurs to some degree in nearly all cases, would have had time to show itself and to permit of a more accurate diagnosis of the distribution and degree of the lesion.

If the palliative treatment be systematically employed there would seem to be no disadvantage in waiting a year before proceeding to radical measures, although in certain selected cases a shorter time limit might be chosen (where electric responses show no tendency toward spontaneous improvement after four to six months).

The advantages of waiting lie in the increased size of the operative field, the more definite localization of the lesion, and the lessened danger to life from shock and hemorrhage.

The clinical demonstration that there is to be a permanent lesion in any given case means that there must be a cicatricial lesion preventing complete nerve regeneration, as has been proven in the consideration of the pathology of the condition.

Here then, as in other traumatic lesions of the peripheral nervous system, the only rational treatment lies in the excision of the damaged tissue, with following nerve suture.

There have been but few secondary operations upon the brachial plexus. Excision of the lesion in a case was advised several years ago (Starr), but owing to the extent of the lesion found the operation was abandoned.

In 1900 Kennedy operated upon a case in which the brachial plexus was involved in a cicatrix causing total palsy of the arm. An operation for the relief of the nerve from adhesions resulted in a practically complete recovery fourteen months after operation. The same author reported operations upon three additional cases of birth palsy in February, 1903. Case I. made a nearly complete recovery in nine months after operation. Case II. five months after operation showed improved electric reaction, but no return of voluntary power. Case III. only three months after operation showed returning faradic response. He recently reported three additional cases, but the time was too short for results to have appeared.

OPERATIVE TECHNIQUE (A. S. TAYLOR).

The patient is anæsthetized and brought to the table with the field prepared for operation. A firm cushion is placed beneath the shoulders, the neck is moderately extended, and the head held toward the sound shoulder. This position gives a good exposure of the operative field.

The incision passes from the posterior border of the sternomastoid

muscle, at the junction of its middle and lower thirds, downward and outward to the clavicle at the junction of its middle and outer thirds. After the skin, platysma, and deep fascia are divided, the omohyoid muscle is exposed near the clavicle, and lying beneath it are the suprascapular vessels. These structures may be retracted downward, or, if the case requires the extra room, the omohyoid first and then the vessels, between double ligatures, may be divided. The transversalis colli vessels are encountered a little below the middle of the wound and divided between double ligatures.

The dissection is rapidly carried through the fat layer to the deep cervical fascia covering the brachial plexus. In all the cases this fascia was thickened at the site of lesion of the nerve trunks. This fascia is divided in the direction of the original incision, is dissected away from the nerve trunks to expose them freely (Fig. 10, 1). The damaged portions of nerve are usually noticeably thickened and of greater density than the normal nerves. The extent and distribution of the paralysis, determined before operation, gives the clue as to which roots are at fault. Usually the junction of the fifth and sixth roots is the site of maximum damage. The thickened indurated areas are determined by palpation and are excised by means of a sharp scalpel. Scissors should never be used for this purpose as they pinch the nerve ends so much as to seriously interfere with their later regeneration.

The nerve ends are brought into apposition by lateral sutures of fine silk involving the nerve sheaths only, while the neck and shoulder are approximated to prevent tension on the sutures. Cargile membrane is wrapped about the anastomosis to prevent connective-tissue ingrowth. The omohyoid muscle, if it has been divided, is sutured with catgut. The platysma and skin are sutured with silk. A firm sterile dressing is applied, and the bandage is made to approximate the shoulder to the head (Fig. 6, Case VI., 2). This position must be maintained for from two to three weeks to permit of firm union between the nerve ends. In the first five cases it was very difficult to maintain this position by bandages, especially during the struggling incidental to recovery from the anæsthetic. It must be remembered that a single moment's separation of head and shoulder after the nerve sutures are tied will spoil the operation. Preceding Case VI. a plaster splint was moulded to the head and paralyzed extremity so as to maintain them in the desired relative position. After hardening it was removed and lined with soft material. At the operation, as soon as the nerve sutures were tied, the cast was slipped on and the skin wound sutured without danger of tension on the nerves (Fig. 6, Case VI., 2). The child could readily be lifted about later without danger to the suture. A neater splint was designed later, with buckles and webbing straps to eliminate the necessity for bandages (Fig. 6, Case VII., 2).

It will be noticed that (Fig. 10, 2) (*a*) the tissues to be excised lie

in close proximity to the phrenic nerve and internal jugular vein, and to the junction of the cervical sympathetic communications with the spinal nerve root. (*b*) The suprascapular nerve comes off from the junction of the fifth and sixth cervical nerve roots, which, as already stated, is usually the site of maximum damage. This nerve is very small in children, but it should be sutured with the greatest care to proximal nerve stump, since it innervates the external rotators of the shoulder, the paralysis of which permits the posterior dislocation of the shoulder often seen in the older cases.

In cases where the lesion is more extensive, especially where it lies beneath the clavicle, a wider exposure is necessary (Fig. 10, 2). It is obtained as follows: The first skin incision is carried downward along the line between the pectoralis major and deltoid muscles, which are then separated. The clavicle is divided in the line of the skin incision by a Gigli saw; the subclavius muscle is divided as are the omohyoid muscle and suprascapular vessels if they have been spared in the earlier steps of the operation. When the outer fragment of the clavicle and the shoulder are retracted outward the entire brachial plexus, with more or less fat in front of it, is exposed down to the upper margin of the pectoralis minor muscle. (If this muscle is divided a still wider exposure is given, but this is seldom required.)

When the nerve suture is completed the divided muscles are repaired, the clavicle is sutured with chromic gut, and the skin closed with silk as before.

This operation, because of its length, the loss of blood, and the shock involved, is applicable only to older children.

Where the lesion involves the whole plexus it would be wise to do only a portion of the repair work at the first operation and complete the repair at a later operation, thus avoiding too long and exhausting a procedure at one time. (See Case 1.)

After-treatment. When the immobilization is removed, three weeks after operation, the measures indicated in Palliative Treatment are systematically employed. As soon as evidence of returning voluntary power appears in the paralyzed muscles, attention should be given to encouraging voluntary motions in all directions, thus educating the motor centres of the brain to control muscles that were up to that time beyond their influence. Improvement, though slow at first, will continue for a number of years until more or less complete recovery occurs.

A definite prognosis as to the amount of recovery that will follow operation cannot be given until several months have elapsed.[1]

[1] In the older cases where contracture deformities are already present the procedures advocated by Whitman (New York Surgical Society, March, 1905) are of great assistance in regaining the usefulness of the limb. The method consists in forcible reduction of the deformities under ether in several sittings, and the maintenance of improvement, so gained by forcible passive motion in the interval, based on the principle of Lorenz's reduction of congenital dislocation of the hip.

HISTORY OF CASES (A. S. TAYLOR).

During the past two years seven cases have been operated upon by the method detailed. Their histories are as follows:

CASE I.—I. H., aged twenty-five months. Patient is a girl who presented complete paralysis of the left arm, as shown in Fig. 6, Case I., 1. A complete lesion of the entire brachial plexus was diagnosticated. She was a first child, vertex presentation. Labor was prolonged and difficult. Instruments were used and strong traction exerted on the head. Complete paralysis of the left arm was noted a few days after birth. No spontaneous improvement resulted during the first two months after birth. After four months, however, slight flexion of the fingers and wrist was possible. In every other way the child was perfectly healthy. There was R. D. in all the muscles of the upper extremity. Contractures produced the deformity shown in the case. An indurated area corresponding to the site of the sixth, seventh, and eighth cervical nerves were found on palpation.

Operation May 18, 1903; chloroform. The more extensive exposure as described above (Fig. 10, 2) was made in this case. The region of the normal brachial plexes was occupied by dense fibrous tissue, in which it was difficult to make out any nerve trunk. After some dissection the proximal ends of the nerves were identified, and the nerves were divided near the outer border of the scalenus anticus muscle. Distally, the nerves were identified and the division made just beyond the cicatricial area, which was in all about 2.5 cm. wide (Fig. 11, Case I.). The nerves were sutured to the proximal stumps with silk, the clavicle was united with chromic gut, Cargile membrane was wrapped around the anastomoses. The wound was closed without drainage. The operation lasted somewhat over one and one-half hours; a moderate amount of blood was lost. The child was returned to bed in fair condition about 3 P.M. Five hours later the temperature was 106°; pulse, 180; respirations, 68. In spite of bathing, rectal irrigation, and stimulation, the temperature rose to 108.8° the following morning. The pulse was imperceptible, and the child died at 10 A.M., about nineteen hours after operation. Cultures taken from the wound immediately after death were sterile and the wound appeared all right.

CASE II.—Annie H., aged ten years. Typical birth palsy of milder grade was present, as shown in Fig. 7, Case II., 1. The patient was a fourth child, vertex presentation, markedly asphyxiated after a dry labor of seven hours' duration. Forceps were used and great force employed in delivery. Palsy of the right arm was at once noticed. At the end of four months improvement began and continued for a year. Slight contractures have occurred in all the muscles paralyzed, producing the characteristic deformity.

Palpation disclosed a cicatricial induration, most marked at the juncture of the fifth and sixth cervical roots.

Operation June 17, 1903; chloroform. The lesser operation was done as described above (Fig. 10, 1). The deep fascia was thick-

FIG. 6



Case I., 1 Case IV., 1 Case V., 1.

Case VI., 1. Case VII., 1 and 2. Case VI., 2.

Case I., 1. Entire brachial lesion of the left arm.
Case IV., 1. Typical laceration brachial birth palsy of the right arm.
Case V., 1. Mild degree of laceration brachial birth palsy of the right arm.
Case VI., 1. Mild degree of laceration brachial birth palsy of the left arm.
Case VI., 2. Shows the attitude maintained, without a moment's intermission, for three weeks after operation. The picture was taken at the end of three weeks.
Case VII., 1. Typical laceration brachial birth palsy of the right arm.
Case VII., 2. Shows the perfected cast for maintaining the proper postoperative position.

ened in front of the nerve lesion, which involved the fifth and sixth nerves, of which the damaged portion (about 1 cm.) was excised (Fig. 11, Case II.). The nerves were sutured and the wound closed as usual. The operation lasted one hour. Very little blood was

FIG 7. Case II



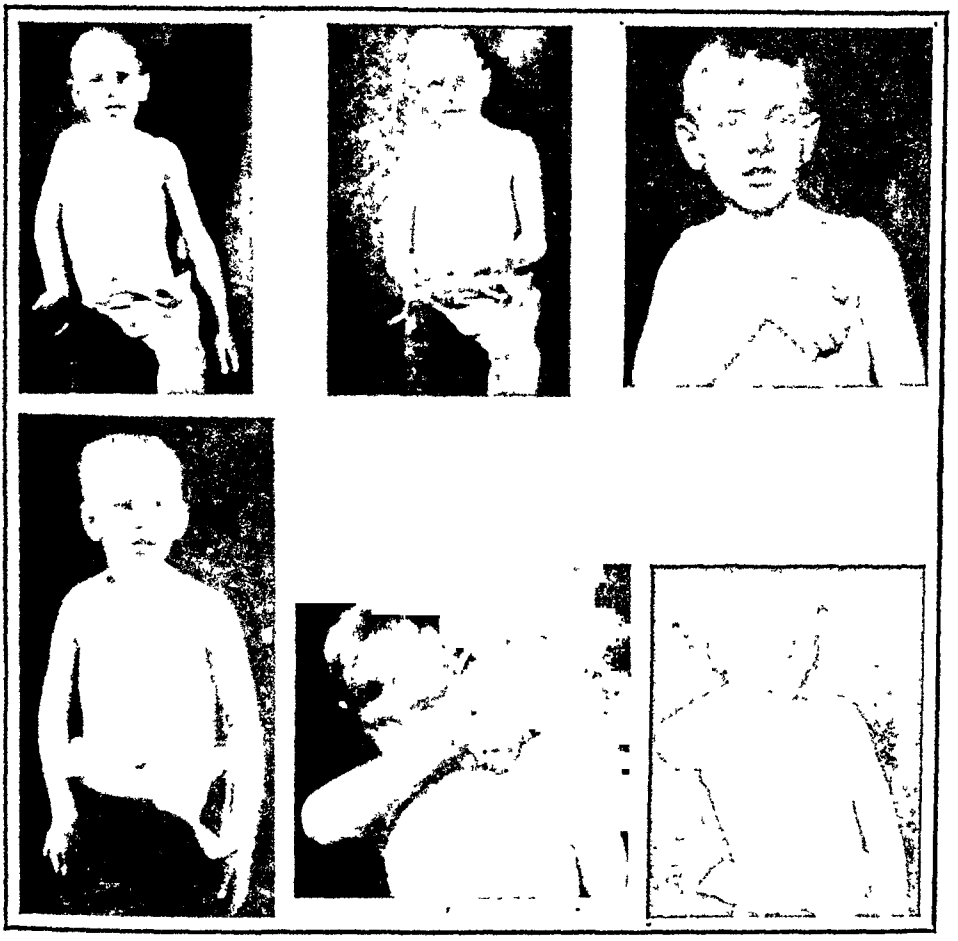
1 (Typical laceration brachial birth palsy.) Ordinary position in which the patient held the right arm before operation.
2 The amount of possible supination of the hand before operation
3 Amount of elevation of arm possible before operation
4 Showing patient's ability to place the hand on the top of the head eighteen months after operation (Compare with 3)
5 Showing patient's nearly normal ability to elevate the arm eighteen months after operation.
6 Showing the amount of supination of the right hand eighteen months after operation

lost. There was no reaction. The wound healed by primary union. After three weeks the dressings were removed and the limb left free. Marked return of power began in the third month and has continued to the present time. (Compare Plate II., Case II., Figs, 1, 2, and 3 with Figs. 4, 5, and 6.) These show the improvement in range of

muscle action. The muscle power in the distribution of the fifth and sixth cervical nerve roots has been fully doubled.

CASE III.—F. G. C., aged eight years. Birth palsy of the right arm, as shown in Fig. 8, Case III., 1. The habitual position charac-

FIG. 8.—Case III.



1. (Typical laceration brachial birth palsy.) Ordinary position in which patient held right arm before operation.

2. Amount of supination of the right hand possible before operation.

3. Showing extent of muscle power in the biceps and deltoid before operation.

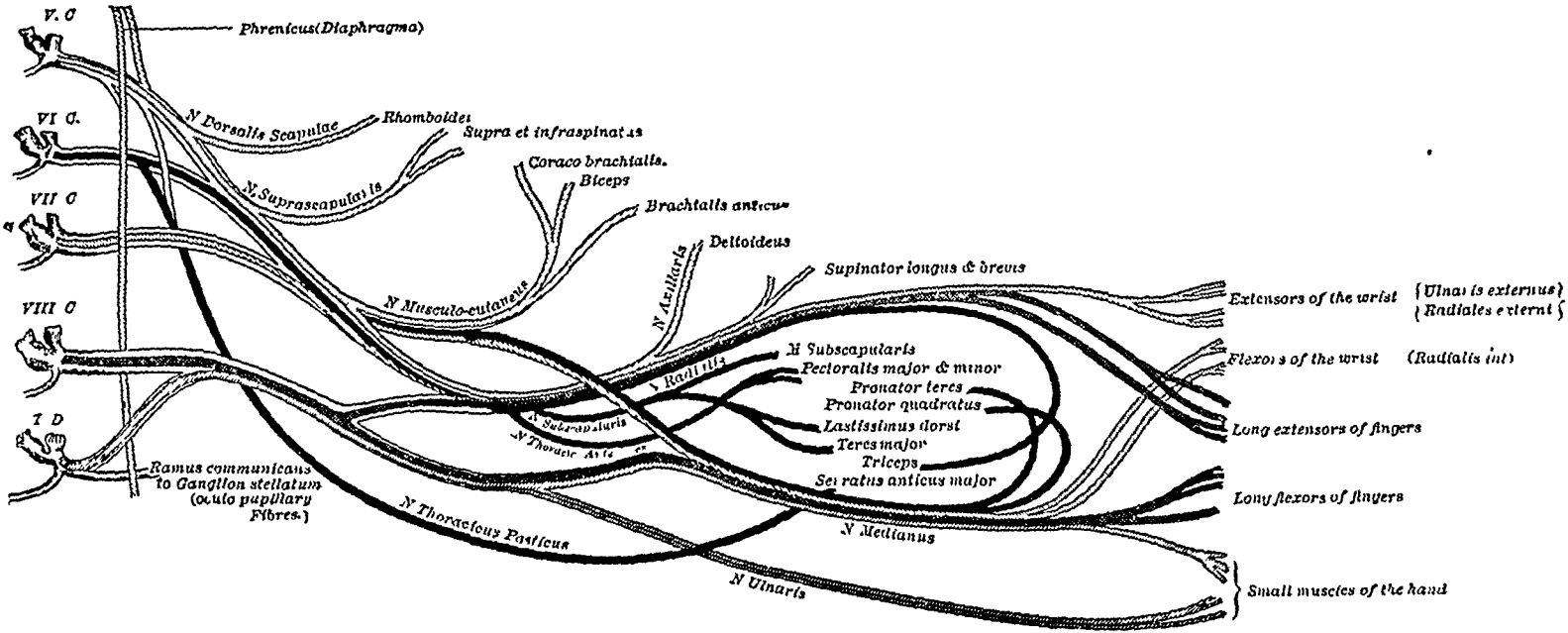
4. Showing great reduction in deformity of the right arm nine months after operation. (Compare with 1.)

5. Showing extent of muscle power in the biceps and deltoid nine months after operation. (Compare with 3.)

6. Showing still greater return of muscle power ten months after operation. (Compare with 3 and 5.)

teristic in this lesion. He was the third child (the two preceding confinements were normal), vertex presentation, dry labor, lasting fourteen hours, in which instruments were finally used. After prolonged and forcible traction he was born asphyxiated and with total

Fig 9

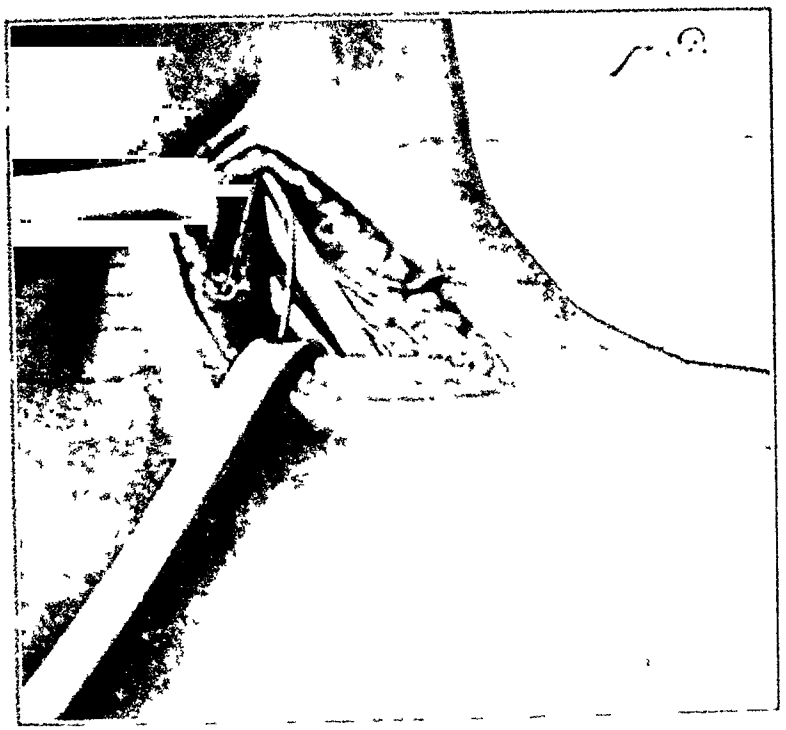


Semidiagrammatic scheme (Kocher) to show the formation of the brachial plexus and the nerve supply to the muscles of the upper extremity.

Fig. 10.

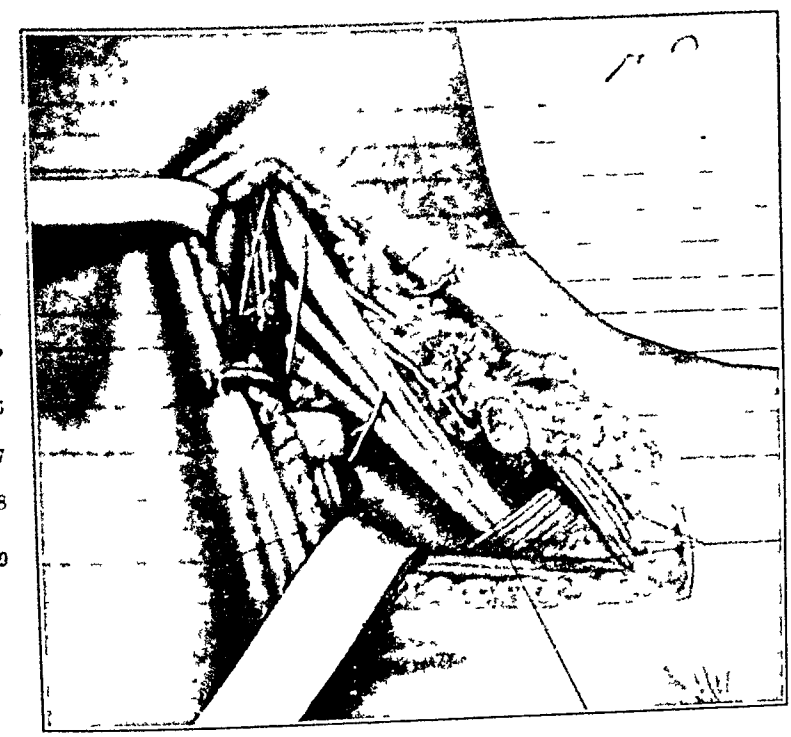
Reproductions from original drawings of dissections of the operative field.

1



1, scalenus anticus muscle; 2, phrenic nerve; 3, internal jugular vein; 4, transversalis colli artery divided; 5, VII cervical root; 6, [illegible] root; 7, V cervical root; 8, scalenus medius muscle; 9, VI. cervical root; 10, transversalis colli artery; 11, suprascapular nerve; 12, nerve to subclavius muscle; 13, clavicle; 14, nerve to scalenus anticus muscle.

2



1, phrenic nerve; 2, scalenus anticus muscle; 3, internal jugular vein; 4, transversalis colli artery; 5, omohyoid muscle, divided; 6, suprascapular artery, divided; 7, VIII. cervical and I. dorsal roots; 8, external anterior thoracic nerve; 9, subclavian artery; 10, V. cervical root, 11, VI. cervical root; 12, scalenus medius muscle; 13, nerve to scalenus anticus muscle; 14, suprascapular nerve; 15, transversalis colli artery; 16, VII cervical root; 17, omohyoid muscle, divided; 18, suprascapular artery; 19, clavicle and subclavius muscle, divided and retracted, 20, deltoid, pectoralis minor, pectoralis major (muscles); 21, nerve to subclavius muscle.

paralysis of the right arm. During the first year there was some slight return of power, but there has been no improvement since that time. The right upper extremity and half of the shoulder girdle are distinctly undeveloped compared to the left side, as shown in Fig. 8, Case III., and the *x*-ray pictures. Palpation of the lesion shows the fifth and sixth nerves involved in a cicatricial mass. There is loss of reaction to the faradic current and R. D. in all muscles of the fifth and sixth distribution. The shoulder is a flail-joint. The elbow cannot be extended nearer than 30 degrees to a straight line. The wrist is markedly flexed and ulnar adducted. The extremity is cooler than the normal one and the hand is purple colored. The bones of the damaged extremity are from 12 to 20 per cent. shorter than the corresponding bones on the opposite side. There is a pronounced posteroinferior dislocation of the shoulder. The coracoid and acromion processes are longer, more pointed, and bend more downward and forward than normal.

Operation June 24, 1903; chloroform. The simpler procedure was resorted to; the fascia in front of the plexus was much thickened and adherent to the nerves. When the plexus was exposed, a most interesting condition was found; the external nerve division derived from the junction of the fifth and sixth cervical roots was torn away and displaced downward and inward 2.5 cm., and was adherent to the front of the scalenus anticus muscle by firm fibrous tissue. The nerve was dissected free and its damaged end removed. The junction of the fifth and sixth nerve and the posterior division derived from them was the seat of cicatricial induration. The damaged tissue was excised (Fig. 11, Case III.), the freshened ends sutured, Cargile membrane used, and the wound closed as usual. The operation lasted one hour. Primary union resulted. After three weeks the extremity was allowed free mobility. The recovery was complicated by a severe bronchitis lasting two weeks. The return of power began decidedly at the end of the fifth month. At the end of the ninth month the patient was able to move the arm as shown in Fig. 8, Case III., 5. One month later, as shown in 6. He continues to improve steadily, but slowly.

Case IV.—Joseph B., aged eight months. Typical birth palsy (Fig. 6, Case IV., 1). He was the third child. The two preceding labors were normal and easy. Vertex presentation. Labor seventy-two hours, conducted by a midwife, who, when the head was born, pulled forcibly upon it to deliver the shoulders, which were caught. Two days later the paralysis of the right arm was noticed. No improvement occurred up to the time of operation. The child was small, frail, poorly nourished, and had diarrhœa; no signs of other disease. He was kept in the hospital for two months, until his diarrhœa had disappeared and his general condition had very greatly improved.

Operation July 22, 1903. Cocaine 1 per cent. and adrenalin

1:4000. As the dissection in this case would be slight, it was thought that cocaine and adrenalin would avoid the risk of a general anæsthetic. The mixed solution was injected into the skin, which after

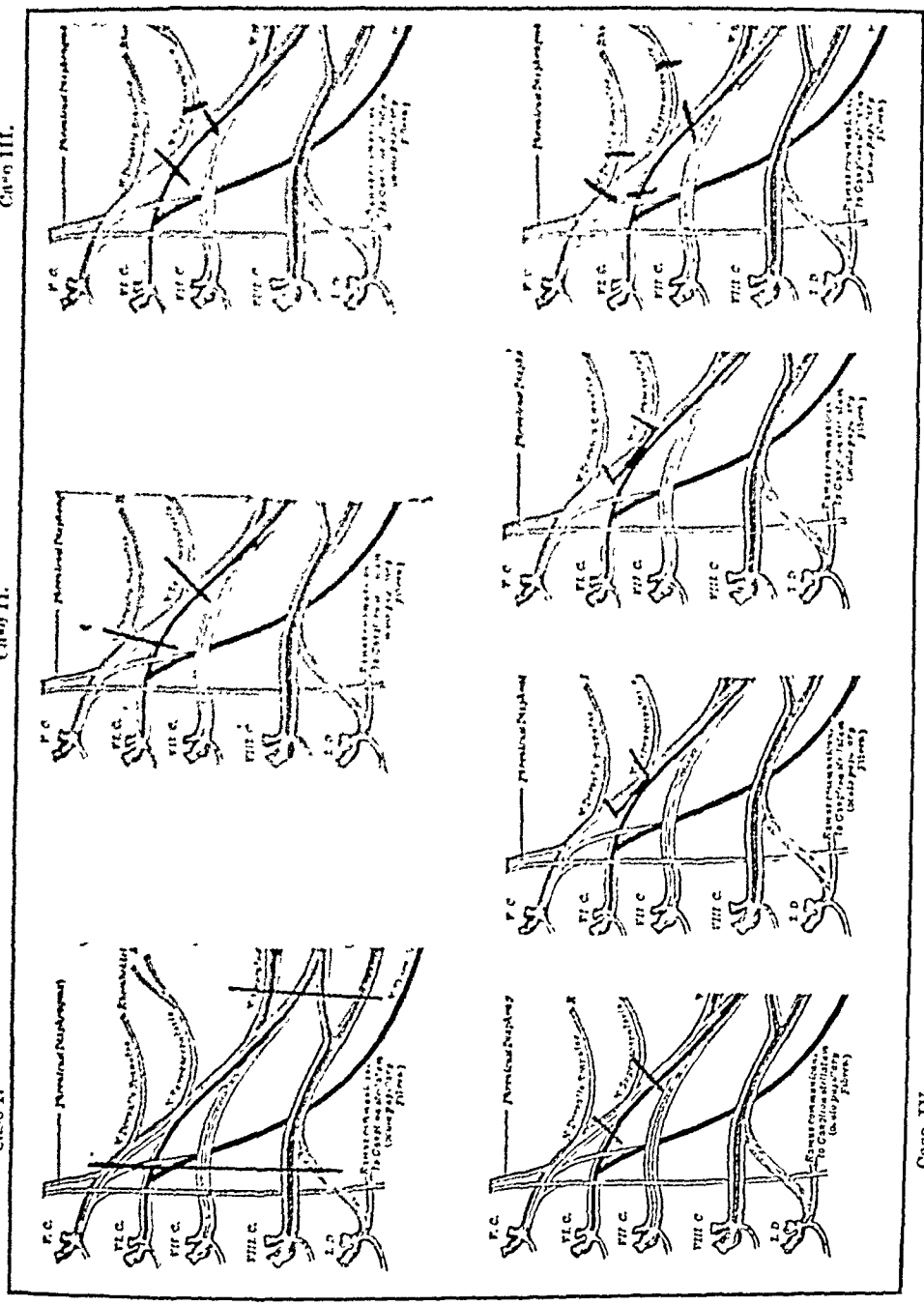
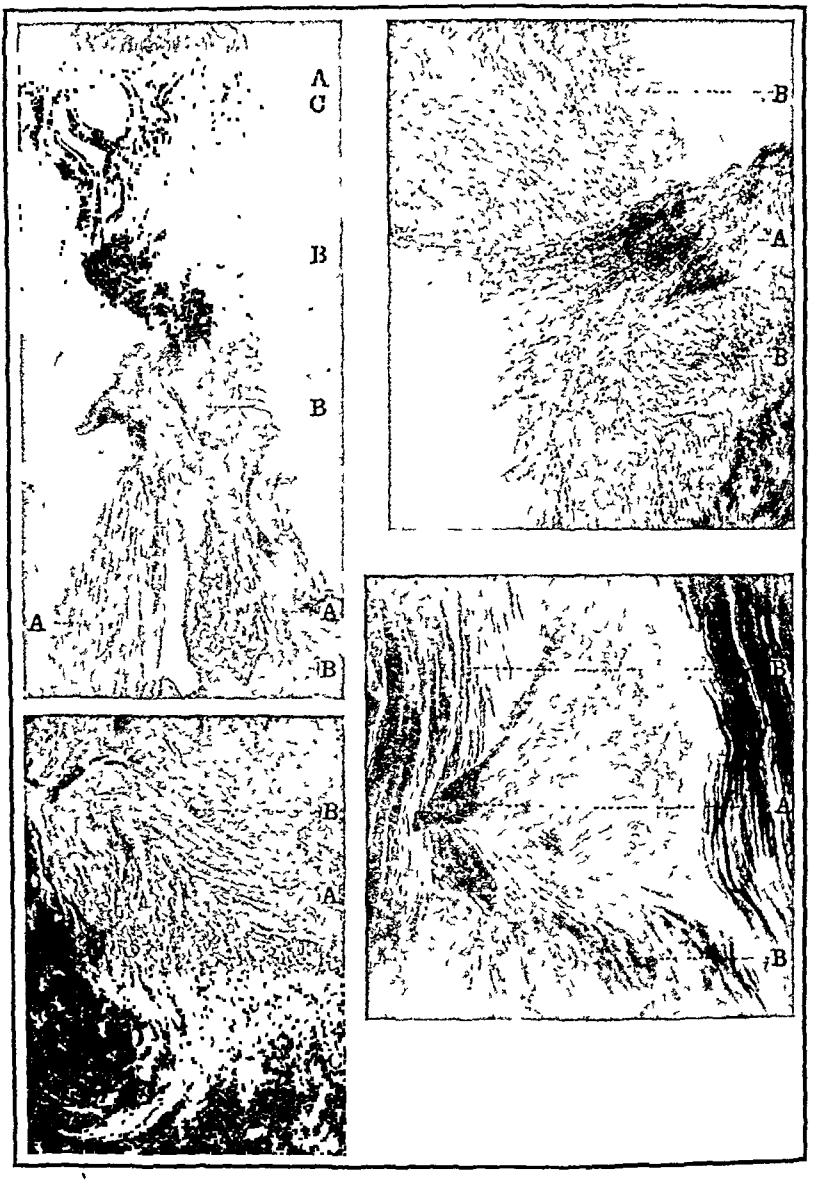


FIG. 11

Diagrams representing in a schematic way the location and amount of nerve tissue removed in each case. The portion removed lies between the black lines drawn across the nerves involved.

five minutes was divided, and the dissection carried quickly through the fat to the plexus. The cicatrix was localized at the junction of the fifth and sixth nerves and the overlying fascia. The solution

FIG. 12



3 4

1 Longitudinal section through piece excised from case *A*, degenerated nerve fibres; *B*, hemorrhages in the perineural sheath, *C*, buckling inward of perineural sheath upon the nerve strands

2 Band of connective tissue, *A*, lying across nerve bundle *B*

3 Kink, *B*, in nerve fibres, *A*, which terminate in connective-tissue mass, *C*, containing pigment (blood clot)

4. Buckle in perineural sheath, *A*, which compresses the nerve fibres. Broken-up remains of the myelin sheath shown at *B*.

was injected into the fifth and sixth nerves proximally. After five minutes the cicatrix was excised (Fig. 11, Case IV.) and the operation completed as before. The operation lasted fifty minutes, and with very slight loss of blood. The child was returned to bed in fair condition, although the temperature had risen to 103°; pulse 154; respiration 80 during the operation. A few hours after the operation the diarrhœa, which had been absent for a month, recurred. The temperature remained 103° continuously and the child was very restless. In spite of treatment the temperature mounted slowly. On the third day there was suppression of urine and the child died. Cultures from the wound were sterile and there were no complications in the chest.

CASE V.—George H., aged four and a half years. Second child. Vertex presentation. Very difficult labor. Instruments used after nineteen hours and "great pulling" was employed. The right arm was paralyzed at birth. Movements began to return at the end of three months, but after the end of six months improvements ceased. Paralysis, deformity, contractures, and atrophy typical of a medium grade of birth palsy, as shown in Fig. 6, Case V., 1, resulted. Electric reaction and palpation showed that the junction of the fifth and sixth nerves was the site of lesion.

Operation May 17, 1904; ether. The usual operation was done, and the fifth root from near the spine down to and including some of the junction of the fifth and sixth roots was removed (Fig. 11, Case V.). The sutures and closures were done as usual. Operation lasted thirty-five minutes. There was no reaction. Primary union resulted. The function of the muscles supplied by the fifth and part of the sixth cervical roots remained completely in abeyance for the first three months. Slow improvement has occurred since, until at the end of six months about two-thirds of the former muscle power and range of movement had returned. This child was very refractory and has followed none of the instructions as to after-treatment, and therefore the best results are not to be hoped for in his case.

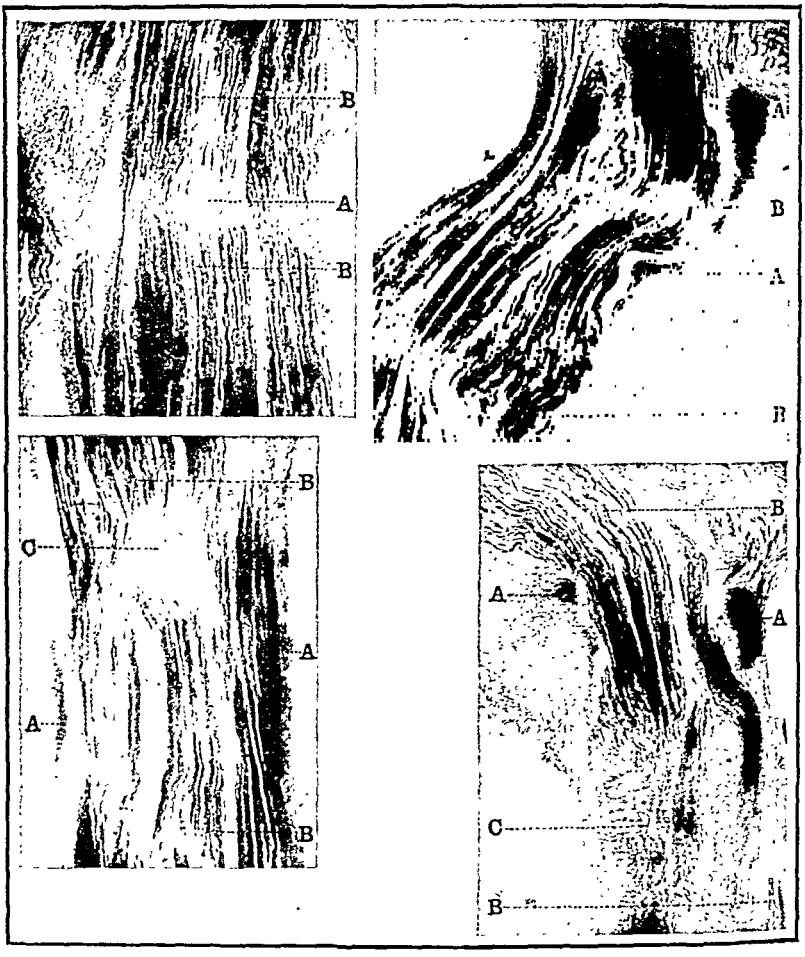
CASE VI.—Rose B., aged eleven years. First child. Vertex presentation. Delivered by a midwife, who pulled on the head. Partial paralysis of the left upper extremity was noticed within the first two days. After several months improvement began and continued for about two years, appearing first in the hand muscles and then extending upward. Finally, improvement ceased and the patient now has a typical birth palsy of mild degree (Fig. 6, Case VI., 1).

The nerve lesion could not be made out by palpation in this case as in the preceding ones, but was located by the type and range of palsy at the junction of the fifth and sixth cervical root, in the upper and outer part of the fifth cervical root principally.

Operation November 12, 1904; ether. The usual operation was done. The fascia lying just in front of the nerve roots was much thickened, was adherent to the front of the scalenus anticus muscle

and to the fifth and sixth nerve roots, from all of which it was dissected away. The lesion was found in the fifth root and in the outer part of the junction of the fifth and sixth root. The damaged

FIG. 13



1. Area of compression, *A*, showing degenerated nerve fibres above and below, *B*.
2. Buckling inward of perineural sheath and pigment deposit at *A*. Torn nerve fibres, *B*.
3. An area of extreme compression: *A*, pigment deposit in perineural sheath (old hemorrhage; *B*, broken-up myelin sheath; *C*, torn and compressed nerve fibres.
4. *A*, pigment, the remains of an old hemorrhage in perineural sheath; *B*, fragments of myelin in degenerated nerve fibres; *C*, wavy bundles of torn and degenerated nerve fibres.

tissue was excised (Fig. 11, Case VI.) and the operation completed as usual. Operation lasted thirty minutes. There was no reaction and the wound healed by primary union.

In this case and the succeeding one sufficient time has not elapsed for the return of power.

CASE VII.—Edward A., aged sixteen months; fourth child. Labor was difficult; version was performed and the case was terminated as a breech. He weighed eleven pounds. Paralysis of the right arm was first noticed at the end of a week, and involved the whole extremity, which assumed the typical attitude. After about three months improvement began in the muscles of the hand and forearm, but finally ceased at fourteen months, leaving him with a typical birth palsy (Fig. 6, Case VII., 1). An induration about 1 cm. in diameter was easily palpable beneath the skin at Erb's point on the right side. He was a sturdy, well-nourished child.

Operation November 19, 1904; ether. Usual operation. The deep fascia was found thickened in front of the nerves and adherent to them. On dissecting it away carefully it was found that the fifth root had been completely torn across, proximal to its union with the sixth root. The proximal end was bulbous, displaced to and adherent to the anteroexternal part of the scalenus anticus muscle. The distal end was bulbous, was adherent to and covered the sixth root. The suprascapular nerve was fibrous for a distance of almost a centimetre from its origin. The damaged tissue was excised (Fig. 11, Case VII.), the nerve sutured, and the operation completed as usual. Operation lasted forty-five minutes. The maximum postoperative temperature was 100° (twelve hours after operation). Primary union resulted.

RESULTS (A. S. TAYLOR).

Mortality. It will be noticed that of the seven cases the first and fourth died (about 30 per cent.).

The first case was a well-developed, healthy blonde girl two years old. The operation lasted one and one-half hours; the loss of blood was moderate; all the roots of the left brachial plexus were divided at the outer edge of the scalenus anticus muscle. The clavicle was divided. This operation was of some magnitude.

The patient left the table in good condition, but within a few hours the temperature rose rapidly and steadily, as did pulse and respirations, until death occurred within twenty hours. (Temperature, 108.8°; pulse, over 200 and almost imperceptible; respirations, 68.) Cultures from the wound were sterile.

Unquestionably shock entered materially into this unfortunate result, but there was much in the clinical picture outside the realm of shock which closely identifies this case with those victims of "lymphatic diathesis," "acetonæmia," and similar little-understood conditions, in whom death occurs without reason even after trivial operations.

Infection does not enter into the problem, since the wound was dry and clean and cultures were sterile.

The fourth case was a poorly nourished child, which was in the hospital several weeks suffering from diarrhœa. During the month preceding operation there was no diarrhœa and the weight increased steadily. Immediately after the operation diarrhœa recurred in aggravated form; there was suppression of urine on the third day and the child died.

In this case death was due to the diarrhœa and the suppression of urine, but these unquestionably were precipitated by the operative procedure.

The other five cases recovered without the least evidence of shock or reaction. Even in Case VII., only sixteen months old, although the operation lasted forty-five minutes under ether anæsthesia, considerable manipulation was necessary and the fifth and sixth roots were divided, the maximum postoperative temperature was 100°, and there was no evidence of shock.

In spite of the two unfortunate cases in this series, there is every reason to believe that the mortality in a large number of cases will be very slight. Because of the complete absence of reaction in the other five cases it would seem as though the two deaths were not entirely due to the operative procedure.

Functional Improvement. The return of function in a divided and sutured nerve occurs only after months, and the longer the time elapsed between injury and operation the slower, relatively, it returns.

In cases of several years' duration there will probably never be a development of limb equal to that of its opposite fellow. This could not be expected. But the disparity (due to growth and even hypertrophy from overuse on the sound side) between the two will either not increase at all or at least will increase in a less rapid ratio.

Only in Cases II. and III. has sufficient time elapsed to give definite results. In Case II. the nutrition of the arm was good before operation. The increased range of motion is indicated in the photograph (Fig. 7, Case II.). The muscles are decidedly stronger than they were.

In Case III. the increased range of motion is shown by the photographs (Fig. 8, Case III). The extremity, instead of being cold and purplish, is now warm and of normal color. The flesh is firmer and the muscles stronger. In the shoulder, instead of a flail-joint, there is still a complete posterior dislocation, but the humerus is pulled well upward by the deltoid. The reduction of the dislocation is prevented by the distorted coracoid and acromion processes, as well as the contractures in the anterior soft structures.

He can now reach his face and head with his right hand and also button his clothing, a thing which he had never been able to do before operation.

Case V. is beginning to show return of power.

It is worthy of note that in Cases II. and III. operation was performed ten and eight years, respectively, after the injury, and yet regeneration with return of function is occurring. This is strong evidence in favor of the rule that nerve suture should be done regardless of the time elapsed since injury, provided there is muscle tissue left to be acted upon through regenerated nerve.

CONCLUSIONS.

This research embodies new facts in the etiology and pathology of the laceration type of birth palsy, the development of the relationship thereto of the symptomatology, and the establishment of a scientific basis for treatment.

1. The cause of the laceration type of birth palsy is tension on the nerve trunks, which first ruptures the nerve sheath and then the nerve fibres. The prevention of this serious lesion of the cervical nerve trunks rests with the obstetrician, who should not overstretch the child's neck in the process of delivery.

2. The persistence of the palsy is clearly explained by the pathological findings, viz.:

(*a*) Rupture of the perineural sheath with hemorrhage into its substance, resulting in the formation of hæmatomata or hæmatomatous infiltration into the neighboring tissues.

(*b*) The cicatricial contraction following organization of the blood clot and repair of the rent in the perineural sheath. The connective tissue thus formed indents and presses upon the nerve bundles, strangulating them and preventing regeneration of the nerve fibres. In some instances the same result is accomplished by the turning inward of the perineural sheath upon the nerve bundles.

3. The nature of the lesion in all cases demands excision of the damaged areas and suture of the divided ends as soon as it is proven that spontaneous repair will not take place. The plan of treatment is then the same as that for peripheral nerve injuries elsewhere.

4. In *all* cases such treatment as will prevent contractures and deformities and maintain muscle tone in the paralyzed limb should be systematically used until either spontaneous recovery occurs or operation is done. (Traumatic neuritis is a contraindication to active treatment.) It is obvious that the above measures should be continued after operation.

5. The proper time for surgical interference is not yet definitely fixed. It appears, however, to be much later than two or three months after birth, as advised in Kennedy's report. At the present time one year would seem to be a reasonable delay before operation.

6. Sufficient time has not elapsed in the majority of the cases in this series for final results to have appeared.

At the end of eighteen months in two cases (II. and III.) the improvement in nutrition, range of motion, and muscle power in the paralyzed limb have been sufficient to demonstrate the value of the operative procedure.

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REVIEWS.

THE VERMIFORM APPENDIX AND ITS DISEASES. By HOWARD A. KELLY, A.B., M.D., Professor of Gynecology in the Johns Hopkins University, and E. HURDON, M.D., Assistant in Gynecology in the Johns Hopkins University, Baltimore. With 399 original illustrations, some in colors, and 3 lithographic plates. Philadelphia and London: W. B. Saunders & Co., 1905.

WHEN first glancing at this extensive work on appendicitis one is at once struck with the value of the illustrations, which are wonderfully clear and true. In fact, the illustrations tell the whole story of the appendix and its diseases, depicting its anomalies, its pathological lesions and the various methods of treating them. In no work which we have seen have the illustrations been better or so elaborate.

The work is most complete and deals with every aspect of the subject. The opening chapters have to do with the history of appendicitis and are most interesting. On reading this portion of the book one is surprised to find that long before appendicitis was generally understood and its proper treatment instituted, isolated authorities here and there over the world had described very clearly cases of appendicitis, and some of these had even recommended the proper treatment. It is evident that but for the conservatism of certain eminent authorities the advance which was made in this line of surgery after the publication of Fitz's paper in 1886 might readily have taken place nearly a generation before. In the early history of appendicitis three names stand out in this narrative: Mélier (1827), Volz (1846), and Lewis, of New York (1856). To Fitz, of Boston, however, the author gives the greatest credit.

Following the section on the history there comes the portion dealing with the anatomy of the appendix, about which it is unnecessary to say more than that it is complete and that the illustrations are deserving of especial commendation. The physiology of the appendix is given considerable space and is dealt with in a very entertaining manner. The chapters devoted to the pathology of the appendix are probably the most valuable in the book, and not the least instructive is that chapter dealing with the subject of thrombosis and its results. In this portion of the work, and also in that dealing with the symptoms and treatment, the text contains frequent brief sketches of cases which illustrate and elucidate the authors' statements. The etiology of appendicitis, which is a

matter of such general interest at the present day, is very fully and interestingly discussed. It is true that the authors are unable to furnish any altogether satisfactory explanation of its great frequency at the present day, but all the various theories in regard to this question are set forth.

The chapters on the symptoms of the various forms of appendicitis are very complete. The authors' classification of the varieties of appendicitis is, we think, very satisfactory, and its universal adoption would render reports and statistics much more comprehensive. Excellent chapters on appendicitis in typhoid fever and appendicitis in children are to be found.

The portion of the book devoted to treatment, although it may be open to the criticism of some individual surgeons, on the whole expresses the consensus of opinion of American surgeons. We are glad to see an explanation of that so frequently misunderstood term "interval operation." Kelly explains that in acute appendicitis it is never right to wait for the interval, but the interval operation should be performed when the patient has passed through an acute attack. The subject of operative technique is dealt with and all the various methods of operating for this disease and its complications fairly presented. Considerable space is given to the subject of anæsthesia, in which nitrous oxide is highly extolled.

The reference to the College of Physicians of Philadelphia as "The College of Physicians and Surgeons" in one part of the book and in another as "The College of Surgeons" is to be regretted. This is all the more noticeable because Kelly has the honor of Fellowship in this old institution.

Among the latter chapters every possible complication and sequela of appendicitis is thoroughly described. Neoplasms, specific infections, and the medicolegal aspect of appendicitis are all amply discussed.

The reader is struck throughout this exhaustive treatise with the thorough way in which the literature of the subject has been studied, and made use of, and is impressed with the enormous amount of work that this has entailed. There can be little doubt that this book contains the most complete and exhaustive study of appendicitis in all its phases that we have. J. H. G.

A Text-book of the Practice of Medicine. For Students and Practitioners. By Hobart Amory Hare, M.D., B.Sc. Philadelphia and New York: Lea Brothers & Co., 1905.

This most recent text-book of medicine promises to be as well received and as justly popular as its distinguished author's previous successful works on diagnosis and therapeutics. Dr. Hare's long

experience as a teacher and writer, combined with his extensive clinical work, has rendered him particularly able to write a book which would combine all the elements so essential for both the practitioner and student of medicine. This is especially noticeable in the sections of the work which deal with treatment. As would be expected, these are most exhaustive, interesting, and authoritative. We have never met with a medical text-book which was so thoroughly satisfactory in its method of handling the treatment of diseased conditions and in which the therapeutic side of the medical art was so well expounded. The author recognizes that the treatment of disease is, after all, the main object of the physician, and his work fully meets all requirements in this regard. In the portion dealing with the pathology and diagnosis of diseased conditions the reader is impressed with the author's clear and lucid expositions, and cannot fail to appreciate their value. One feature of the book which deserves especial commendation is the very important contribution on the subject of tropical diseases by Major Kiefer, U. S. A. There is an excellent section on diseases of the nervous system, in the preparation of which the author had the collaboration of Dr. Pickett. The illustrations with which the book abounds are well chosen and executed. The arrangement of the letter-press is such as to render the work easy of reference and to add greatly to the value of the work. J. N. H.

On Becoming Blind: Advice for the Use of Persons Losing Their Sight. By Dr. Emile Javal, Honorary Director of the Ophthalmic Laboratory of the Ecole des Hautes Etudes, Member of the Academy of Medicine. Translated by Carroll E. Edson, A.M., M.D. New York: The Macmillan Co. London: Macmillan & Co., Ltd., 1905.

The impression left by the reading of this book is a sad one. The precepts therein contained will accomplish much to mitigate the calamity so far as may be.

Perhaps the greatest of the many misfortunes which blindness entails is loss of liberty. The central idea of Dr. Javal's work is based upon this fact; practically all the directions he has to give relate to extending the blind person's sphere of action. The home should be a model of regularity, every article should always be in its proper place so as to be readily recovered; the clock's place in the room should always be the same; its ticking will then furnish correct orientation to the sightless. The first advice to be given an adult who loses his sight is to learn Braille. In spite of its exceeding slowness, reading and writing Braille are the principal resource for hours of solitude, though the use of a special typewriter has come to replace manual writing to some extent.

The blind are particularly apt to suffer from lack of physical exercise. Home gymnastics are very tiresome. Dr. Javal highly recommends a tandem tricycle.

While opposed to marriage between blind persons, the writer does not think that marriage is necessarily contraindicated if only one of the couple is blind. The fear of blindness becoming hereditary is very slight. He concludes, however, that before marrying the blind will do well to have the subject considered twice by some devoted friends.

The book is full of useful suggestions, frequently going into minute details, but the importance of these to the blind is very great. He advises that the blind should not wear gloves, as they interfere with the sense of touch. Plates for eating may be divided in the bottom by a ridge to separate the meat from the vegetables; but to pick the bones from the fish or to put the proper amount of mustard upon the meat is nearly impossible. These acts should be done for the blind by a servant or companion. Many of the directions are simple enough, but as the writer rather pathetically remarks, why has no one thought of putting them down.

This book is not to be read for pleasure; but it is a most useful one for all who are brought in contact with the blind, most so for those who are compelled to witness this great calamity in the person of some member of their family. T. B. S.

DISEASES OF THE HEART AND AORTA. By THOMAS E. SATTERTHWAITE, M.D., Professor of Medicine in the New York Post-Graduate Medical School; Consulting Physician to the Post-Graduate, Orthopædic, and Babies' Hospital; President of the Medical Association of the Greater City of New York. New York: E. R. Pelton, 1905.

THIS book is much to be commended, for it is not too lengthy and yet presents in a remarkably clear and practical manner not only the essentials but many other original and practical points of value. The author has drawn freely on his own wide experience, and details numerous cases illustrative of particular conditions.

The chapters on syphilis of the heart and on cardiac displacements are especially interesting. The functional diseases are fully discussed, and a chapter is devoted to the consideration of Graves' disease.

Particularly valuable are the author's remarks on the general management of heart diseases, and on the Nauheim methods with American modifications. The illustrations, while not numerous, are good. F. F.

CLINICAL DIAGNOSIS AND URINALYSIS. The Medical Epitome Series. By JAMES RAE ARNEILL, A.B., M.D., Professor of Medicine and Clinical Medicine, University of Colorado, etc. Series edited by VICTOR COX PEDERSEN, A.M., M.D., Instructor in Surgery and Anæsthetist at the New York Polyclinic Medical School and Hospital, etc. Philadelphia and New York: Lea Brothers & Co., 1905.

THIS volume presents in abbreviated form the subject of laboratory diagnosis as compiled by the author from the standard textbooks and from his own observations, and it covers practically all the important points.

There is some fault to be found in the arrangement of the subject matter, especially in the opening chapters dealing with general considerations and with hæmatology. The chapters devoted to the examination of the gastric contents are excellent and entirely practical, and the same may be said of the examination of the sputum.

In the discussion of the feces the chemistry is purposely omitted. Although, as the author states, the chemical examination of the fæces is usually of little value, the tests for so-called "occult" blood are certainly worthy of consideration as an aid to diagnosis in doubtful cases.

Under "Miscellaneous Examinations," exception may be taken to the brief and casual manner in which lumbar puncture is disposed of. The subject of urinalysis is covered thoroughly, and in an up-to-date manner.

The illustrations, with the exception of the colored plate supposed to represent Tallquist's color scale, are very satisfactory.

F. F.

A TREATISE ON PLAGUE: DEALING WITH THE HISTORICAL, EPIDEMIOLOGICAL, CLINICAL, THERAPEUTICAL, AND PREVENTIVE ASPECTS OF THE DISEASE. By W. J. SIMPSON, M.D. Aberd., F.R.C.P. Lond., D.P.H. Camb. Cambridge: University Press, 1905.

THE book is dedicated "to all those who are actively interested in plague and its prevention," but so engrossing is the work that one feels after reading a few chapters that the dedication must have been meant for him alone. The author's close association with the plague during the epidemic in Cape Colony and in Calcutta and Hong Kong lends a personal note to the descriptions which give the book a peculiar individuality and charm. The story of the disease, one of such great historical interest and present-day importance, is told with much accuracy and care. The first 225 pages; about one-half the treatise, are devoted to "The History and Distribution of the Plague and the Epidemics of Plague." The history

of the early epidemics, the account of the plague in India and the description of the present pandemic are given very fully, and these chapters abound with notes to literature and with quotations from the earlier writers. A very good description of the "Plague in the Individual" follows. This part is divided into several chapters on the "Morbid Anatomy and Pathology," "Channels of Infection," "Clinical Features," "Diagnosis and Prognosis," and "Treatment." Autopsy protocols and illustrative cases aid in giving an accurate idea of the disease as it is seen. Finally, the last part, covering something over 100 pages, is devoted to "Measures for Prevention and Suppression of Plague."

The book, from beginning to end, abounds in interest and points of practical importance. Maps, tables, temperature charts, and photographs help to illustrate the text. Though there are microphotographs of the plague bacillus it is rather disappointing that the author has not inserted drawings to illustrate the pathology of the disease. But one who reads this treatise cannot fail to become intensely interested in the subject. W. T. L.

WHARTON AND STILLE'S MEDICAL JURISPRUDENCE. Vol. II., Poisons. By ROBERT AMORY, A.M., M.D., and ROBERT L. EMERSON, A.B., M.D. Fifth edition. Rochester, N. Y.: The Lawyers' Co-operating Publishing Co., 1905.

THE general scope and character of this large treatise is so familiar, and the work has remained as a standard for so many years, that a detailed description is quite unnecessary. In fact, the vast amount of material which it contains almost precludes review. A very extensive and fourth revision was made in 1884, the first since 1873, and the present is the fifth edition. Much revision, of course, has been made to bring the material up to the present date, and in large part the text has been recast or rewritten. This has been done very successfully and many new subjects have been inserted, as, for example, the chapter on amyl alcohol poisoning, the discussions on poisoning by sulphonal, and the evil effects of acetanilid and the other coal-tar derivatives. Ptomain and toxalbumin poisons are also considered at some length. The newer biological tests for blood are discussed and numerous references are made to the modern work dealing with chemical problems concerned in toxicology. Occasionally one finds certain delinquencies in regard to modern methods of diagnosis, between cases of poisoning, and well-recognized diseased processes. Few criticisms, however, can be made, and, altogether, the volume is a veritable storehouse of information for both physician and lawyer. W. T. L.

THE DETECTION OF POISONS AND STRONG DRUGS, INCLUDING THE QUANTITATIVE ESTIMATION OF MEDICINAL PRINCIPLES IN CERTAIN CRUDE MATERIALS. By DR. WILHELM AUTENRIETH, Professor in the University of Frieberg. Authorized translation from the third enlarged German edition by WILLIAM H. WARREN, Ph.D., Professor of Chemistry, Medical Department of Washington University, St. Louis, Mo. Seventeen illustrations. Philadelphia: P. Blakiston's Son & Co., 1905.

THIS book is a very well-arranged and clear treatise, presenting methods for the detection of the more common poisonous substances. Although its general plan closely follows that of a manual of chemical analysis, the style is expanded sufficiently to make it easily read. The descriptions are very clear and readily understood. Several methods of qualitative and one of quantitative analysis are usually briefly given.

The various poisons are divided into three groups, and considerations of these groups constitute the first three chapters. The fourth chapter, on poisons not in the three main groups, includes a brief account of the methods of detecting poisons in blood and the biological tests for human blood. The last chapter deals with the quantitative estimation of alkaloids and other drugs in crude materials. This subject, usually omitted in American works of this kind, has apparently reached more importance in Germany, and this part of the book should be especially useful.

The book contains about 220 pages. As a laboratory guide it is sure to prove very useful. G. C. R.

THE URINE AND FECES IN DIAGNOSIS. By OTTO HENSEL, Ph.G., M.D., and RICHARD WEIL, A.M., M.D., in collaboration with SMITH ELY JELLIFFE, M.D., Ph.D. Illustrated with 116 engravings and 10 colored plates. Philadelphia and New York: Lea Brothers & Co., 1905.

LARGE text-books and treatises upon clinical diagnosis, books in which all the excretions and secretions of the body, as well as the blood, receive consideration, are numerous and well known; but it would be difficult to find a small volume covering just the ground contained in the present book. The microscopy and chemistry of the urine has been written of so often that the subject does not afford much chance for originality. The study of the feces has, however, been much neglected until quite recently, and it is with great satisfaction that one finds a book half of which is devoted exclusively to this important subject. In many obscure conditions

a careful and systematic microscopic examination of the feces clears a doubtful point or makes a diagnosis, while, as recent work has shown, no little value must be accorded to the chemical examination in investigations upon metabolism.

The first 150 pages of the book are devoted to urinalysis, and the treatment is not strikingly different from what one finds in many other text-books upon this subject. It is, however, very good, concise, and aims at the practical side of the question. In every detail the book is modern. One finds descriptions of all the newer methods of chemical examination, especially those which have been found most useful and applicable to the clinical laboratory; while older and impracticable methods handed down from text-book to text-book have wisely been omitted. Fortunately the original references are quoted. Over 150 pages are devoted to examinations of the feces. There are full chapters upon the macroscopic and microscopic examination, an excellent *résumé* on the bacteriology of the intestinal tract, taken almost entirely from Ford's work on the subject, chapters on the chemical examination with methods for the determination of total nitrogen and fat, the characteristics in disease, and the animal parasites. One might wish, perhaps, that the parasitology had been more fully discussed. Though most of the illustrations are quite good, a few are very inefficient. Such an important cut as the one representing the ova of uncinaria duodenalis ought to have been much better.

The book, however, is really excellent, and will be of great use to student and practitioner. W. T. L.

THE NAKED-EYE ANATOMY OF THE HUMAN TEETH. By THOMAS E. CONSTANT, Licentiate of the Royal College of Physicians, London; Licentiate in Dental Surgery and Member of the Royal College of Surgeons of England. Bristol: John Wright & Co. London: Simpkin, Marshall, Hamilton, Kent & Co., Ltd., 1905.

THE ignorance of medical men generally regarding the teeth is often noticed by dental surgeons and not infrequently in surgical clinics, and often causes unnecessary suffering on the part of their patients. We are inclined to believe that the medical profession would be better off for the perusal of this volume, which, however, is strictly anatomical and has nothing to say of those diseases for which the patient consults the physician and which yet have their origin in defective teeth.

This small book is especially designed for the use of dental students and fulfils its purpose admirably. Many of the illustrations are new and beautiful, but some of those relating to the anatomy of the mouth and neck are quite familiar. J. H. G.

ADDRESSES AND OTHER PAPERS. By WILLIAM W. KEEN, M.D., LL.D., F.R.C.S. Illustrated. Philadelphia and London: W. B. Saunders & Co., 1905.

DR. KEEN has put the medical profession more than ever in his debt by gathering together some of his contributions to medical literature and publishing them in book form. They cover many subjects. The first address is entitled "The Early History of Practical Anatomy." It is quite profusely illustrated with interesting and rare plates, and although not very lengthy, it constitutes a strong evidence of the erudition of the author. The second address will be read with especial interest by all the members of the profession who received their education in Philadelphia. It is a "History of the Philadelphia School of Anatomy and its Relation to Medical Teaching." This school, which flourished for over half a century, exerted a great influence on medical science in America. As Dr. Keen says, "it is only necessary to point to the thousands of men who have studied and dissected here, and here begun their scientific lives, and are now spread all over the country, and in fact all over the world, doing the best of work as practitioners, teachers, writers, and original investigators." It was not only an anatomical school; within its walls Dr. Da Costa gave private courses on physical diagnosis and did much experimental work. S. Weir Mitchell had his physiological laboratory in it, giving there the first purely experimental course on physiology ever given in Philadelphia, and in it began many of his most important physiological researches, and Dr. Hayes Agnew for many years taught anatomy.

It is a great pity that Dr. Keen's introductory delivered at the Commencement of the Woman's Medical College of Pennsylvania, entitled "Our Recent Debts to Vivisection," cannot be as universally circulated among the laity as the ridiculous literature which is disseminated by the opponents of vivisection. In this connection it is a pleasure to note that the author does reprint an article entitled "Vivisection and Brain Surgery," which appeared in *Harper's Magazine*, June, 1893. These two articles alone would make this book a valuable gift to any opponent of vivisection.

Another article which is reprinted from *Harper's Magazine* is "Recent Progress in Surgery." It illustrates Dr. Keen's happy faculty of presenting medical subjects to lay audiences which is also instanced in the article entitled "The Progress of Surgery in the Nineteenth Century," reprinted from the *New York Sun*. This article was one of a series published by that newspaper apropos of the beginning of the twentieth century, and sums up the indebtedness of the human race to the profession of surgery. There are a number of other addresses printed in the volume; all of them are of interest and value. We cannot better conclude than by again presenting our thanks to Dr. Keen and our congratulations to the medical profession on the publication.

F. R. P.

A Laboratory Guide in Elementary Bacteriology. By William Dodge Frost, Ph.D., Assistant Professor of Bacteriology, University of Wisconsin. Third revised edition. New York: The Macmillan Co., 1903.

The book is designed as a guide to a course in bacteriology such as is given in the more advanced medical schools. It is composed of a well-arranged series of experiments which carry the student from the simplest technique to the more complicated bacteriological analyses. The fact that three editions have appeared in as many years shows that it has proved of use.

While some teachers undoubtedly prefer to use such a book with their classes, it seems to us that its employment perhaps detracts from the individuality of the teacher, and makes the student's work more mechanical than it might otherwise be. In planning a course of bacteriology, however, or for independent work by a novice without a teacher, the book can be highly recommended.

G. C. R.

La Rhinoplasties. Par Ch. Nelaton et L. Ombildaune. With 391 illustrations. Eight vo., 430 pages. Paris: G. Steinheil, editeur, 1904.

The volume comprises two parts, one dealing with the deformities and the other with destructive lesions of the nose, copiously illustrated with diagrams remarkably clear and easily understood. The first part treats of congenital malformations, recent and ancient traumatisms, the various operations for the relief thereof, as well as one chapter on hypertrophic acne of the nose. The destructive lesions are discussed in Part II.: first, historically; second, etiologically; and third, types—viz., (*a*) total loss of the nose; (*b*) subtotal loss (the integument and septum completely destroyed, the alæ completely or incompletely destroyed; (*c*) destruction of the base—bone and cartilage involved or destroyed.

In a very exhaustive review the following chapters give in detail the general technique of the anæsthesia, preparation of the patient, the various prosthetic apparatus, transplantations and the rhinoplastic operations depending on the employment of a pedicle of skin—or skin and bone—to replace the site of the original lesion.

The methods are exhaustively discussed and splendidly illustrated by diagrams and pictures from life (author's cases) under the headings: 1. Indian. 2. French. 3. Italian methods. The authors adopt the Indian method as including all those operations in which only a single flap of skin is used from the forehead, not including as do the Germans those methods requiring the use of a

flap from the cheeks. Under this heading the original of this operation among the Koomas as well as those of Graefe, Lisfranc, Langenbeck and others less familiar are discussed. The French and Italian methods include all the other operations recorded and give in detail the operations for the restoration of the various parts—viz., the alæ, the septum, cartilaginous, and cutaneous, as well as the various methods of support by plates of celluloid, hard rubber, metal plates, amber, etc. Under the heading of "Destruction of the Bony Skeleton" are discussed the various injection methods and apparatus, as well as an exhaustive review of the various autoplastic operations from Dieffenbach (1823) to the present day. The restoration of the tip of the nose as well as the secondary operations necessary to improve the contour of the nose after the usual contraction of the primary operation are also thoroughly gone into and discussed. A valuable feature is a criticism of the various operations at the end of each chapter and instruction as to the method to be chosen in each individual case. The work ends with a chapter on correction by means of apparatus. E. A. H.

THE PHARMACOPŒIA OF THE UNITED STATES OF AMERICA. EIGHTH DECENNIAL REVISION. By authority of the United States Pharmacopœial Convention, held at Washington, A.D., 1900. Revised by the Committee of Revision and published by the Board of Trustees. Official from September 1, 1905. Philadelphia: P. Blakiston's Son & Co.

THE new edition of the United States Pharmacopœia contains several important innovations. Probably the most striking of these is the introduction of synthetized products of definite composition which are in common use by the medical profession, the identity, purity, and strength of which are known. This does not apply to compounds of which the composition or mode of manufacture are kept secret, or which are patented. It is also to be noted that trade names are not given in the Pharmacopœia. But one serum remedy is introduced, and that is the antitoxin for diphtheria. Two animal products are introduced, namely, those made from the thyroid and suprarenal glands. Especial attention is directed by the Committee of Revision to the following changes: "The strength of tincture of aconite has been reduced from 35 per cent. to 10 per cent., and that of tincture of veratrum from 40 per cent. to 10 per cent. The strength of tincture of strophanthus has been increased from 5 per cent. to 10 per cent. These changes have been made in order to conform to the standards adopted by the International Conference on Potent Remedies held at Brussels in September, 1902, the object being to make uniform the strength of potent remedies in all parts of the world." F. R. P.

ENLARGEMENT OF THE PROSTATE: ITS HISTORY, ANATOMY, ETIOLOGY, PATHOLOGY, CLINICAL CAUSES, SYMPTOMS, DIAGNOSIS, PROGNOSIS, TREATMENT, TECHNIQUE OF OPERATION, AND AFTER-TREATMENT. By JOHN B. DEAVER, M.D., Surgeon-in-Chief to the German Hospital, Philadelphia, assisted by ASTLEY PASTON COOPER ASHHURST, M.D., Surgeon to the Out-patient Department of the Episcopal Hospital; Assistant Surgeon to the Orthopædic Hospital and to the Dispensary of the German Hospital. Illustrated with 108 full-page plates and a colored frontispiece. Philadelphia: P. Blakiston's Son & Co., 1905.

THIS book is a counterpart of that on the appendix by the same author, and from a book-making point of view is open to the same criticisms. It is light and the print is large, but the uneven and uncut leaves are a decided objection.

The first part of the work deals in an interesting manner with the History of Enlargement of the Prostate Gland. We are inclined to think that rather too much credit is given to Mr. Freyer for the origination of the present method of suprapubic prostatectomy, although no one can deny that he has done much to popularize the method. Various theories regarding the causation of enlargement of the prostate are carefully presented. The author is inclined to the view, so strongly set forth by Ciechanowski, and later confirmed by the investigations of Greene and Brooks and Crandon—viz., that a preceding inflammatory condition of the prostate is the most potent cause of enlargement.

In the section dealing with the treatment the hygienic, di tetic, and constitutional treatment are carefully outlined. Deaver believes that every patient should have a trial of catheterism before resort to prostatectomy is made, although he states plainly that the catheter can produce a cure in no case. The method of using the catheter is carefully set forth and anyone undertaking the treatment of prostatic hypertrophy by this means will do well to study this section of the book carefully. The matter is gone into in great detail, and what is said may be taken as a most reliable guide. The Bottini operation is advised in but one class of enlargement of the prostate —that in which the prostate is small and sclerosed and where its removal by prostatectomy even in otherwise healthy patients would be extremely difficult. A marked preference is shown for the suprapubic route in nearly all operations upon the prostate, either radical or palliative. Although showing this preference, Deaver states that hard, small, fibrous prostates are best removed through the perineum. He believes that the large, soft, adenomatous prostates should never be removed through the perineum. In combating the statement of those preferring the perineal route that the prostate is extravesical, one is hardly prepared to agree with the author that the prostate is no more extravesical than the appendix is extraperitoneal.

The closing chapters deal with the technique of the various methods of prostatectomy. The steps of the operation are very thoroughly and satisfactorily illustrated.

The thorough and painstaking work of Ashhurst is apparent throughout the volume. We have no hesitancy in heartily recommending this work as a guide in the treatment of all forms of enlargement of the prostate. J. H. G.

OBSESSIONS ET LA PSYCHASTHENIE. Par F. RAYMOND et PIERRE JANET. Paris: Felix Alcan, 1904.

RAYMOND and Janet have presented at great length in this volume of 537 pages a clinical and psychological study of the psychical manifestations of the fatigue neuroses. The subject-matter is presented in the form of clinical demonstrations, continuing the style made popular in France by Charcot in his Lecons du Mardi. The psychological analysis by Janet is especially thoroughly and worthy of careful attention.

Formerly the subject-matter of this nature has been treated under the subject heading of neurasthenia. The obsessions and phobias of different kinds have long since been considered as symptoms of the neurasthenic state. The cerebral type of neurasthenia, the predominance of the mental symptoms in the fatigue group, as differentiated from the spinal group or physical fatigue manifestations, has long since been abandoned by those who have given careful study to the subject. The cerebral and spinal exhaustion symptoms cannot be so differentiated, and both groups depend upon the perversion of function of the central nervous system as a whole. The psychical perturbation may dominate the clinical picture in some cases, just as the visceral introspection and physical fatigue does in others. The decrease of the will power to its almost complete abeyance, the perversion of psychical associated action, the domination of the individual by the ideas associated with this disturbance —this is the psychasthenia of Janet. The value of the separation of this group of cases from the general function group is open to question; the tagging of it with a special name complicates the subject and leads to a certain amount of confusion to the minds of those who have not given especial study to the subject. There is no question, however, of the value of the studies presented in this volume to those whose duty it is to treat such cases with an understanding of the underlying mental processes. The impression made by the volume is that the authors have made much out of little, and said it at great length. This, however, is the custom in the Charcot school to present in large and impressive volumes subject-matter which would attract more attention if a certain discrimination in case citation and a judicious elimination of unnecessary detailed discussion were attended to. D. J. McC.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Congenital Icterus with Chronic Splenic Tumor.—v. KRANNHALS (*Deutsche Arch. f. klin. Med.*, 1904, Band lxxxi., No. 596). Minkowski in 1900 drew attention to a remarkable condition of hereditary jaundice associated with urobilinuria, splenomegaly, and siderosis of the kidneys. The instances he cites could be traced through three generations affecting at least eight members of the one family. None of the individuals observed showed any changes in the blood, while the general health was not impaired. v. Krannhals has had under his care three members of a family exhibiting similar symptoms, and from information obtained from one of these, a sister of the mother of the other two, he constructs a family tree. The grandparents of the informant had two children, a son, her uncle, who was yellow, and a daughter, her mother, who had a grayish complexion and splenic tumor; her uncle had one daughter, who was of a grayish-yellow complexion. Her mother had three daughters, of whom one died in infancy; the oldest was yellow and had a splenic tumor, as well as the informant. The oldest daughter had six children. The first pregnancy ended in an abortion. The second child had a grayish-yellow color and distended abdomen and died in infancy; the third, a boy, one of the patients, is yellow and has a splenic tumor; the fourth, a boy, who died at twenty-one years of pulmonary tuberculosis, was yellow and had a splenic tumor; the fifth, a boy, is said to be white; the sixth, a boy, one of the patients, is yellow and has a splenic tumor. The informant had one abortion and two children, who died shortly after birth, both of these being white.

In the cases under observation the stools were always colored, usually firm, never alcoholic; the urine acid, dark red in color, free from albumin, sugar, blood, and hæmoglobin; urobilin was almost constantly present, but never any bile. The blood serum showed bile. The number of red cells was below normal, 2,800,000 and 3,500,000, respectively, in the

men, and on one occasion as low as 1,000,000 in one of the women. The hæmoglobin varied from 55 to 65 per cent. The red-blood corpuscles were regular in contour, varying in size from 6 to 10μ, a few measuring as much as 12μ; there were no nucleated reds, but well-marked polychromatophilia. In at least 2 of the cases the icterus was definitely present from birth.

Cases similar to these have been reported by Hayem, Gilbert, and Bettman. In a number of the French observations the jaundice was only doubtfully congenital and the spleen not enlarged, and in some there was evidence of hepatic disease. As practically nothing is known of the changes at the bottom of the condition, the classification must remain uncertain. The only autopsy on record is taken from Minkowski on a case dead of pneumonia. No special changes were found in the liver, and the gall passages were quite clear. A small pigment stone was found in the gall-bladder. Even microscopically there were no striking changes in the liver. The intestines contained abundant bile. The spleen was enlarged, and microscopically there was simple hyperplasia and hyperæmia. The appearance of the kidneys was striking. Along the cortex there was a peculiar brownish discoloration produced by a granular pigment deposited in the epithelial cells of the convoluted tubules. Not only did this pigment give an intense reaction for iron, but there was a large amount of the metal united with proteids, which gave the reaction only after boiling with ammonium sulphide. From the ash of one kidney about 0.5 gm. of pure iron was obtained. Similar pigmentation did not occur in any other organ. Minkowski concludes that the condition depends upon some peculiar anomaly in the blood pigment transformation, perhaps consequent upon a primary change in the spleen. In Bettman's case the intensity of the jaundice displayed marked variations deepening after excesses in food and drink, after physical excitement, especially anger, after overexertion, and particularly after exposure to cold. Experimentally hæmoglobinuria could be produced by the application of cold, and Bettman assumes an abnormal tendency of the corpuscles to give off hæmoglobin to the serum, thinking that this explains best the variations in the extent of the jaundice and splenic swelling.

The Diagnostic Significance of Strangulation Marks on Round-worms.—W. EBSTEIN (*Deutsche Arch. f. klin. Med.*, 1904, Band lxxxi., p. 543) says that round-worms are remarkable for their wandering propensities, and also for their ability to force their way through very narrow openings. They have been found in the Eustachian tube and in the tear-ducts, and in an instance reported by Bruneau one had reached the external ear by way of the Eustachian tube and through a perforation in the ear drum. Not uncommonly, too, they enter the bile passages, and they have been found in the gall-bladder, and even in the ducts within the liver itself. Worms entering the common duct may not only cause obstructive jaundice, but, by carrying infectious material from the intestine, they may give rise to a suppurative inflammation of the ducts and liver, with metastatic abscesses in other organs.

Ebstein refers to a case previously reported from his clinic by Mertens. The patient, very ill with symptoms of obstructive jaundice and sepsis, began to improve immediately after the passage of two partly macerated round-worms. This striking relation of the improvement to the evacua-

tion of the worms certainly impresses one as more than a coincidence, but Ebstein urges that it establishes only a high degree of probability in favor of the worms or worm having been in the common duct. The remaining doubt, however, he thinks, is quite dispelled by the presence on one of the worms of a strangulation mark situated about 5 cm. below the head. An eminent zoölogist who examined the specimen declared that such markings never occurred normally, and was of the opinion that it could only have been produced by the worm having been struck by its front end in a small canal. This view was supported by the marked difference in diameter of the worm before and behind the constriction, a difference of 1.5 mm. Ebstein thinks that the finding of these strangulation marks may be of diagnostic importance in establishing without doubt the causative relation of round-worms to bile-duct symptoms which may have been present before their passage.

Family Tabes.—CRONER (*Berliner klin. Wochenschrift*, 1904, No. 49, p. 1266) contributes the following instances to show the relation of family to syphilitic disease of the nervous system: In a family of four children where there was apparently no hereditary taint, the three brothers all developed tabes, and the sister died at the age of fifty years of a "paralytic stroke." In another family of four brothers and two sisters, three of the men became tabetic; one brother died young of typhoid fever, one sister of diabetes, and the other sister of miliary tuberculosis. Croner thinks there must be some factors other than chance to determine the point at which the syphilitic virus strikes. Instances like the above might suggest some inherited peculiarity in the cord as an important factor in the etiology of tabes.

SURGERY.

UNDER THE CHARGE OF

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Intertesticular Anastomosis.—GATTI (*Annales des malades organes genito-urinaires*, May 15, 1905, p. 721) defines synorchidism as a fusion of the two testicles, which is an exceptional anomaly, only one case being reported, that of Geoffry St. Hilaire. The two testicles in this case had been welded along the median line, the differential lines were double, and each part of the double testicle received its own particular vessels.

Intertesticular anastomosis, as proposed by Gatti, consisted in making the excretory small canals of the interrupted testicle communicate

by way of the obstructed path, with the tubules on the internal surface of the sound testicle. In this way, through a process of degeneration, the obstructed semen could find an exit through the seminiferous tubules, the network of Haller, the epididymis and differential canal of the unaffected side.

The grafting of the path of emission of one side to that of the other side determines a degree of traumatism, to which the two testicles can respond by the phenomena of degeneration. These phenomena will be in direct proportion to the intensity of the operative traumatism, and will fail entirely when the traumatism is very slight.

When the trauma is sufficient to cause the phenomena, small diffuse zones at and near the line of anastomosis will appear, with normal zones among them, the volume of the organ remaining normal. If one of the small differential canals is delicately isolated from the connective tissue and perideferential sheath and is sectioned, it will not show the phenomena of degeneration.

When the phenomena are not too grave, there will be a proliferation in which all the tissues participate. The epithelial regeneration tends to reproduce the glandular tissue and to create a tubular anastomosis between the glandular substance of the two united organs. This takes place through slender epithelial outgrowths, at first of only one colony of cells, then more, until finally they form cylindrical plugs, which ultimately become canalized.

This epithelial regeneration is preceded and accompanied by an active vascular and nervous regeneration, which by crossing the line of anastomosis puts into communication the vascular and nervous network of the two testicles.

There will be very little proliferation of connective tissue if, without resecting any of the glandular tissue, but with incision and detachment of the tunica albuginea and the true sheaths, the surfaces to be apposed are denuded entirely with a sharp knife. The fusion of the sheath proper at the line of anastomosis must be prevented, since such a fusion would tend to separate the glandular surfaces by a proliferation of connective tissue.

Intertesticular anastomosis, as thus practised, conserves the vitality of the glandular tissue and spermatogenesis with the abundant presence of spermatozoids, and favors the normal movements. This is not only rational, but is supported by experimental proofs.

The Technique of Amputation of the Penis.—JANSEN (*Centralblatt fur Chirurgie*, May 20, 1905, p. 545) calls attention to two important points demanding especial consideration. The contraction of the urinary outlet is best prevented by a ruffle-shaped formation, through the fixation of the urethral wall, which, in consequence of its unyielding character, maintains a permanent unfolding of the orifice, and more especially through a slitting of the lower wall of the urethra backward for about 1 cm. and attaching the urethra to the skin.

A greater difficulty is to form a stump, the orifice being wide enough, which will permit the escape of the urine, without staining the surrounding parts and the clothes. By the methods at present in vogue, from cicatricial contraction, the urethra turns downward and backward toward the base of the penis, so that the patients are often compelled to assume peculiar attitudes in their efforts to keep clean. The psychical

effects are disagreeable, and often sooner or later a harassing eczema of the surrounding skin sets in.

Jansen makes an oval upper flap of healthy skin, and, after ligating the dorsal artery and the veins, divides the erectile bodies transversely, leaving the urethra about 1 cm. longer. Each corpus cavernosum is closed by interrupted sutures introduced vertically, so that a horizontal line of sutures is formed, thus elevating the urethra indirectly. The projecting urethra is then turned upward over the erectile bodies and fixed by a suture, which takes hold of the albuginea of these bodies, and the periurethral connective tissue of the upper urethral wall without involving the mucous membrane. The end of the urethra is then passed through a transverse incision at the base of the long upper flap, and, after slitting the lower wall of the urethra about 1 cm. backward, its edges are sutured to the edges of the opening in the skin-flap. This flap is then turned downward, properly cut, and sutured to the skin on the under surface of the penis. Drainage must be provided for the blood which escapes under the flap, otherwise there will be a tendency to the formation of a hæmatoma. The epispadias, which is produced by the buried stitch, is lessened by the contraction of the cicatricial tissue, the scar being below, and tending to draw the urinary orifice downward. The urinary outlet will thus be finally situated on the end of the stump.

Constriction of the Ureter.—MONSARRAT (*British Medical Journal*, June 17, 1905, p. 1323) reports 3 cases of constriction of the ureter treated by operation, with good results. In the first he dilated the urethra and passed into the right ureter, through a Kelly speculum, a ureteral catheter, which stopped about three-quarters of an inch from the orifice. He then opened the abdomen and exposed the ureter transperitoneally, made a small incision into it, and passed a fine flexible bougie downward. This stopped about one inch from the bladder, and on making some pressure it was felt to be gripped in the channel, so that the ureter could be pulled upward as one can pull the urethra with an instrument tightly gripped by an urethral stricture. The bougie was then removed, the ureter closed, the peritoneum sutured over it, and the abdomen closed. Later the stricture was dilated from the bladder, when something was felt to give way, which supports the idea that the ureter was bound down in the pelvis by adhesions, being the subject of extradural and not intradural stricture.

In a second case operation showed a tumor formed of dilated kidney and pelvis, with the ureter lying on the dilated pelvis for about one and a half inches, and opening by a fine probe-sized orifice into the pelvis about three inches above the lower margin of the pelvis. A small incision was made in the outer wall of the pelvis, and a probe passed into the ureter. The intervening wall was divided and the edges thus made were sutured together on both sides of this cut. In this way the ureter was made to communicate with the pelvis by a long slit-like opening about one and a half inches in length. The final result was very satisfactory.

In the third case the constriction was at the junction of the ureter and pelvis, which was dilated. A fine pair of dressing-forceps was introduced from the pelvis and the stricture dilated. After free irrigation of the pelvis with water, the incision in the pelvis was closed and the wound in the soft parts drained. There was no return of the stricture symptoms.

Some Remarks on Cases Involving Operative Loss of Continuity of the Common Bile-duct.—Mayo (*Annals of Surgery*, July, 1905, p. 90) reports that of 1100 operations on the gall-bladder and bile passages in the hands of C. H. Mayo and himself, 159 were upon the common bile-duct, and of these 7 involved complete loss of the continuity of the common bile-duct as a direct result of the operation; 5 were intentionally produced in the attempt to remove a malignant neoplasm, 1 was caused accidentally, and 1 followed an extensive operation for gallstone disease.

His experience teaches that: first, the common duct may be united end to end by through-and-through sutures of catgut. It is essential that a few supporting sutures should be placed in the surrounding tissues, and that a portion of the circumference of the line of sutures be left open for relief of tension and drainage. Second, the common duct, and in certain cases the hepatic duct, may be implanted into the duodenum, provided a peritoneal-covered portion of the intestine be chosen for the purpose. Third, to facilitate these operations, the second portion of the duodenum should be loosened and drawn to the right and held by fixation sutures, preventing tension on the duct suture line. Fourth, drainage, if necessary, should be pliable, covered with rubber tissue and placed as distant to the suture line as will serve the purpose of protection against leakage. It is a mistake to insert gauze drainage to the suture line. The plastic lymph which should protect the suture line becomes entangled in the meshes of the gauze, and so disturbs the union as to permit, as occurred in one of Mayo's cases, a biliary fistula.

He reports a case in which, by a second operation, the hepatic duct was implanted into the duodenum about three inches from the pylorus, on account of a stricture of the common bile-duct, a result of the first operation, in which the gall-bladder had been removed. A cholecystenterostomy would have been far easier as a second operation, provided, of course, that drainage would have restored its function to a sufficient extent. Since that time Mayo has been more conservative about the removal of the gall-bladder in connection with common bile-duct surgery.

Castration for Sexual Inversion.—Fere (*Revue de chirurgie*, March, 1905, p. 293) says that castration in sexual inversion has been proposed to relieve social inconveniences, to prevent hereditary transmission, and as a cure for the inversion. The descendants of these people are certainly sometimes tainted by degeneracy, but they have in some cases given birth to perfectly normal children. Fere believes that such a radical measure is not necessary, that the tendency of inverts is not strong toward marriage, and can be still further weakened by encouraging them to lead a chaste life. Some of them are capable of conforming to the general laws of conduct. The difficulty in legalizing castration is increased if we cannot show that the operation improves the instincts of the invert, but must confess that it may make his mental condition worse. They often suffer from a consciousness of their anomaly, even to the extent of meditating suicide.

We do not know the lesion which causes the inversion. The testicle has been accused and ablation has been proposed. Fere, in one case, thought that the cause might be due to some lesion in the brain. Castration had been suggested to the patient, who showed a strong inclination to follow this advice, and consulted a neurologist, who advised against it. In a second case castration had been performed. The

testicles were replaced by two foreign bodies of about the same size and consistency as the organs. The patient believed that his aspect and expression had been changed from the sexual point of view. When the wound was healed and the dressings removed he was permitted to feel the prosthetic apparatus. He was shocked, although he had anticipated the absence of the normal sensibility of the organ. Frequent palpation led to irritation, so that it became necessary to remove the foreign bodies. He was distressed at the emptiness of the scrotum, the loss of voice, which he thought indispensable to his virility and intelligence, and the thought that everybody noticed him. He continued to be subjected to the same temptations as before the operation, and he was less capable of resisting them. The inclination to suicide which had worried him before the operation now left him, but he became addicted to the use of opium.

Fere believes that while such a case does not make an absolute rule, it deserves consideration, and should be placed with the evidence to show the history of the physical condition after castration.

Spinal Anæsthesia by Stovaine in Diseases of the Urinary Tract.—HERESCO and STROMINGER (*Annales des malades organes genitourinaires*, April 15, 1905, p. 629) made injections in 11 cases. In 10 they obtained sufficient analgesia to perform the operation. In 1, because of faulty technique, analgesia was not obtained. Their failure to make a greater number of trials is due to the fact that their results did not agree with those of other more enthusiastic observers. They had some accidents, which, although not serious enough to place the patients' lives in danger, sufficiently supported their apprehensions concerning this method.

Résumé of disturbances caused by the injections: In 2 cases the temperature was 38.3° and 38.7°. In 4 cases the temperature was 39.2°, 39.3°, 39.6°, 39.8°. In 6 cases there was slight headache; in 3 nausea; in 2 relaxation of the anal sphincter; in 1 myosis; in 3 weakness of the lower extremities; in only 1 persistent pains with contracture of the upper extremity; in 1 respiration was difficult, painful, and frequent; and in 1 there was insomnia.

Concerning the Treatment of Prostatics with Incomplete Chronic Aseptic Retention and Dilatation of the Urinary Bladder.—NICOLICH (*Annales des malades des organes genitourinaires*, May 1, 1905, p. 667) compares the unfavorable results he obtained in 10 cases of prostatic hypertrophy with retention, treated by antiseptic and successive evacuation of small quantities of urine (9 died), with his results from transvesical prostatectomy (4 cures in 4 cases).

From these results he considered that the latter method of treatment should be adopted. All 4 patients operated on had been made bedridden, probably from the use of the catheter, and had suffered severely, losing much strength. In all there was enormous retention with incontinence. One was in a very bad general condition, and another suffered from heart disease. All 4 recovered without the complications, so frequently associated with catheterization, and after from seventeen to thirty days urination was spontaneous. Such results in those cases formerly considered as *noli me tangere* cases, in the writer's opinion, makes this the treatment of first choice.

After removing the prostate he tampons the cavity with a large quantity of iodoform gauze to compress the vessels which are much dilated in a distended bladder, to ward off infection and to absorb the urine which is coming from the kidneys.

He prefers transvesical prostatectomy, because, according to his experience, it is more easy, rapid, and complete, and is less dangerous than the perineal. He compares with his own the results of M. Panchet's operations in 3 cases, in each of which there was considerable residual urine after operation. The difference is due to the superiority of the transvesical over the perineal operation.

THERAPEUTICS.

UNDER THE CHARGE OF

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ASSISTED BY

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Mercury in Tabes Dorsalis.—DR. MAURICE FAURE states that the preferable treatment is that by injections of the soluble salts, given daily, twice or thrice a week. The injections should be intramuscular, given slowly and followed by massage. The puncture should be at least an inch and a quarter deep. The choice of the salt depends upon the case to be treated. The biniodide, the benzoate, and the bichloride are more active, but less well borne than the cacodylate or the hermophenylate when given in the same dose. In younger patients whose condition is good, the more active salts may be used in large doses. The dose, however, should be calculated for each patient according to the effect produced; when the insoluble salts are given the injection should be in one large dose per week, while the less irritating preparations are better given in repeated smaller dosage. The treatment is given in series of, in case of the insoluble salts, six to twelve injections; in case of the soluble salts, six to forty. These series are given at intervals of proper duration. The number of series in a "treatment" varies from two to four, as also does the number of "treatments" in a year. The patient should be given all the mercury that he will tolerate without exhibiting toxic symptoms, and proper diet, hygiene, and mode of life should be prescribed.—*Journal de médecine de Paris*, 1905, No. 11, p. 113.

Lumbar Puncture in Epidemic Cerebrospinal Meningitis.—DR. L. TOBLER has employed lumbar puncture as a means of treatment in 7 cases of epidemic meningitis. In 3 of these unmistakable benefit resulted. Direct relief of the severe headache was experienced; the patients rested easily, were able to sleep and take nourishment, and the stiffness of the neck and the general hyperæsthesia became less. In 1

case there was a definite fall in temperature. One of the patients proceeded directly to recovery; a second was much better in three weeks, experienced a short recurrence of the symptoms and then recovered; a third, after two punctures, was much improved, and after another puncture recovered. In the fourth case the only benefit obtained was a mitigation of the headache, and the other 3 cases did not improve after the procedure, and ended fatally.—*Correspondenz-Blatt für Schweizer Aerzte*, 1905, No. 7, p. 209.

Lumbar Puncture in Cerebrospinal Meningitis.—Prof. H. Lenhartz has treated 45 cases by this method and has found that it does no harm and frequently may be practised with much benefit. It is better not to draw off more than 8 or 10 drachms at a time. Occasionally one puncture effects a cure; at other times a number are necessary at daily or longer intervals. —*La Semaine médicale*, 1905, No. 13, p. 149.

Specific Treatment of Relapsing Fever.—Dr. Gabritschewsky has determined that this affection develops in the organism substances which are inimical to the living spirochæte, and the accumulation of these substances in the blood of the patient after each relapse has led the author toward the establishment of a serum therapy and diagnosis of this disease. Defibrinated blood from a patient is injected into the circulation of dogs or horses, these animals possessing a natural immunity to the spirochæte, and three weeks later the serum is drawn. Of 20 patients treated with this serum, 12 had but one recurrence of the fever, while 8 had two relapses, which statistics show a great advance over the ordinary treatment of the disease. In serum therapy the second relapse, when it occurs, is after a longer interval, and its duration is shorter.—*Zeitschrift für klinische Medizin*, 1905, Nos. 1 and 2, p. 43.

The Treatment of Tropical Enterocolitis.—Dr. A. Thiroux considers lavage as only a palliation and that laxatives should be used with the greatest caution, constipation, when present, being controllable by means of proper diet. Dietetic treatment is of greatest importance; a mixed diet is preferable, since a strict milk diet tends to cause constipation and flatulence, and an entirely vegetable regimen contains too little nourishment. Among other articles, the author allows fish and shellfish, meat, preferably beef, fruits, and vegetables, except cabbage; beans and peas should be divested of their outer coverings or eaten in the form of purée. The fat of meat should be removed, and butter should be eaten in small amounts only. Alcoholic drinks are not allowed, but coffee may be taken. Cold baths may be employed with benefit, and change of climate often becomes necessary. Spa treatment may at times prove useful.—*Gazette des hôpitaux*, 1905, No. 14, p. 159.

The Oat Treatment of Diabetes.—Dr. S. Lipetz believes that the good results which have been claimed for the employment of oat flour in diabetes are due to the fact that this substance, in spite of its large carbohydrate content, is not absorbed. The assertion that it is useful in combating acidosis appears to be due to the fact that any regulation of the diet increases the tolerance of carbohydrates and tends to diminish acidosis. Usually the dangers of coma can be avoided at times when acidosis is marked by giving large doses of sodium bicarbonate until

the urine becomes alkaline. The author believes it possible that in marked diabetes with alimentary disturbances the use of oat flour may lessen the tendency to intestinal fermentation, and in view of this fact, he advises the employment of small amounts of this substance in all cases.—*Zeitschrift fur klinische Medizin*, 1904, Nos. 1 and 2, p. 188.

The Local Treatment of Obesity.—Dr. F. Allard considers that localized deposits of fatty tissue, such as those that occur in the thoracic or abdominal regions, are amenable to treatment by manual or vibratory massage, by exercises and by the electric current. The hand massage should be *pétrissage*, and the mechanical massage should be performed through the medium of balls or rollers. The employment of a vibrating ball controlled by an electric motor is particularly efficient. Active movements should be confined to the parts involved; the abdomen and hips are most frequently affected, and the movements recommended consist of flexion, extension, lateral movements, and circumduction of the trunk. Various apparatus may be employed, and the exercises facilitate the elimination of the excess of fat, both in the abdominal cavity and its wall; they also improve portal circulation, and consequently favorably affect the digestive function. The faradic current, through a roller electrode, of sufficient intensity to produce muscular contraction and with sufficiently frequent interruptions to tetanize the muscles, is indicated. The treatment of choice for localized fatty deposits is faradization with vibration. The metal vibrator should be attached to one pole of the battery, while a flat electrode, attached to the other pole, is applied to some other region of the body. The current should be regulated, just as when faradization alone is given.—*Revue de thérapeutique*, 1905, No. 6, p. 191.

Hydrochloric Acid in Gastritis.—Dr. F. Weitlauer states that the alkalies are inferior to hydrochloric acid in gastric catarrh. In his opinion, the dilute acid, in doses from 6 to 10 drops in water, is preferable, and acts best in combination with tannic acid in the proportion of 40 drops to 9 grains mixed with 3 ounces of water. Of this mixture a tablespoonful is taken one-half hour after the morning and evening meals in half a tumblerful of warm water. This must be rain- or distilled water, since the salts in ordinary waters are likely to neutralize the hydrochloric acid, and the dose should be taken through a tube, in order to save the teeth. The addition of the tannic acid lessens the tendency of the hydrochloric acid to disturb the stomach and exerts an astringent action upon the catarrhal condition. This treatment is indicated when the patient, several hours after a full meal, is nauseated or experiences a sense of fulness, and also in the declining stage of an acute gastritis.—*Munchener medizinische Wochenschrift*, 1905, No. 8, p. 357.

The Destiny of Carbon Dioxide in the Stomach.—Dr. K. Löning has shown by experiments upon man and animals that carbon dioxide gas taken into the stomach in water is absorbed in considerable quantity. For the first ten or fifteen minutes the absorption takes place rapidly, and after the lapse of this time more slowly until after an hour but little remains. This in alcoholic beverages also is absorbed in considerable quantity by the gastric mucous membrane.—*Zeitschrift fur klinische Medizin*, 1905, Nos. 1 and 2, p. 26.

The Prevention of Puerperal Fever.—Dr. B. Credé believes in the importance of sterilizing the internal genitals after parturition. In order to accomplish this object, all clots should be removed and a suppository inserted into the uterus itself, into the cervix, or merely into the vagina. He recommends a suppository consisting of collargol and talcum powder, of each a grain and a half, cocoa butter, 30 grains. After such a suppository is inserted the vagina should be loosely packed with sterile gauze, which with the suppository may be renewed. The collargol tends to sterile the secretions without harming the tissues. If infection has taken place, douches of 1:2000 to 1:5000 collargol solution are given, with the introduction of a suppository in the intervals of these. In advanced infection the author advocates the intravenous injection of 2 to 2½ drachms of a 2 per cent. collargol solution.—*Zentralblatt für Gynäkologie*, 1905, No. 6, p. 163.

Fibrolysine.—Dr. Felix Mendel states that this combination of thiosinamine and sodium salicylate injected into the veins exercises a solvent action upon fibrous tissues, such as cicatrices, pannus, adhesions of serous membranes, etc. Direct injection into the tissues, as well as intravenous injection, is free from pain and unpleasant after-effects. Pain due to involvement of nerves in scar tissue has disappeared after a single injection of this substance into the cicatrix, but in treating such lesions the injections should be stopped as soon as the hard tissue has regained its normal texture. Further injections tend to reproduce the induration. Ankyloses due to scar contraction are especially amenable to treatment, and good results have been obtained in parametritis, which had resisted other methods. The author considers it possible that fibrolysine may have a field of usefulness in conditions of connective-tissue inflammations, such as occur in cirrhosis and nephritis.—*Therapeutische Monatshefte*, 1905, No. 2, p. 43.

Treatment of Floating Kidney.—Dr. M. I. Afanasieff considers that surgical methods are indicated in floating kidney only when the organ is the seat of malignant disease or other grave complication is present. In most cases it is possible to manage by less drastic measures. The object is to increase the fat of the patient, so that the perirenal fatty tissue may be of sufficient quantity to hold the organ in place. The diet and mode of life prescribed should be the opposite of that indicated in obesity. These means must be supplemented by the use of an abdominal belt. The kidney is replaced by manipulation and kept in position by a pad attached to the belt. This appliance is put on each morning by the patient and laced from below upward, so as to support the kidney. Medical treatment calculated to lessen the nervous state in which these patients are often found consists in the administration of belladonna, atropine, or hyoscyamus in appropriate dosage. Constipation and cardiac conditions may also require attention.—*Deutsche Medizinal-Zeitung*, 1905, No. 15, p. 157.

Diphtheria Antitoxin in Severe Whooping-cough.—Dr. Passalaequa has employed this means of treatment successfully in 7 cases. The action of the serum is usually manifest after one injection, but three or more are necessary to obtain a complete result. The diminution of the number and intensity of paroxysms is soon noticed, and while the

influence of the serum is not so rapidly shown as in diphtheria, it is, as a rule, plain by the sixth day. This form of treatment is especially indicated in cases with bronchial or pulmonary complications.—*Revue francaise de médecine et de chirurgie*, 1905, No. 11, p. 259.

Urotropine in Pyelitis.—Prof. Howard A. Kelly states that urotropine is a most valuable remedy in the prevention of inflammations of the renal pelvis. It is especially effective in cases of infection with the colon bacillus and as a prophylactic in patients with impaired kidneys, which, during the course of acute disease, are likely to become involved. —*Medical Record*, 1905, No. 14, p. 521.

Silver Iodide in Urethritis.—Drs. E. H. Siter and A. A. Uhle have used this silver salt in a number of cases with uniformly good results. In their opinion, its points of advantage are that it does not stain and is less irritating than the nitrate; it is not proprietary, and its exact strength can be controlled. Under its use the *ardor urinæ* disappears within twenty-four hours and the discharge within a week. No discomfort attends the injection of the drug in 5 to 10 per cent. strength. Silver iodide is insoluble, consequently it must be used in suspension, preferably in mucilage of quince-seed. It has also been employed in cystitis and ophthalmic conditions with good results.—*University of Pennsylvania Medical Bulletin*, 1905, No. 3, p. 89.

The Treatment of Leg Ulcers.—Dr. Vorner reports excellent results from the treatment of leg ulcers and their causative factors, eczema, and varicose veins by means of elastic roller bandages. Two are used and they should be of good length and firm; the first is snugly applied to the limb, beginning at the base of the toes, and over this the second is fitted. The proper degree of tightness can be learned by experience. The patient wears the dressing all day, but may loosen it at night, in order, if necessary, to apply other treatment. After the symptoms have disappeared the bandages should be worn for some time; then one is removed and finally the other; no recurrences of the lesions have been observed. In certain cases the immovable bandage, rendered so by a mixture of gelatin, 10 per cent.; glycerin, 5 per cent.; boric acid, 5 per cent.; gum tragacanth, ½ per cent., may be used. The mixture being applied to the outside of the first bandage and covered by the second, the ankle-joint being left free. Such a dressing may be removed by wetting with hot water.—*Munchener medizinische Wochenschrift*, 1905, No. 8, p. 350.

Local Anæsthesia in Rectal Operations.—Dr. J. R. Pennington prefers a solution consisting of beta-eucaine lactate, 3 grains; sodium chloride, 11½ grains; solution adrenalin chloride (1: 1000), 10 drops; distilled water, 3½ ounces to cocaine solutions or to sterile water injections. The solution is prepared by mixing and boiling the ingredients, except the adrenalin, which should be added when the mixture is cooled to about 100°. The entire quantity may be injected, and under its influence operations may be performed upon hemorrhoids, external, internal, thrombotic, or cutaneous polyps, prolapsus, fistula, fissure, abscesses, condylomata, etc.—*Journal of the American Medical Association*, 1905, No. 14, p. 1093.

Local Anæsthetics in Rhinolaryngology.—Dr. G. Finder considers that beta-eucaine possesses certain advantages over cocaine, in that its solutions may be boiled, keeps for a long time, and are much less toxic. He has not shown that a mixture of adrenalin and cocaine lessens the toxicity of the latter, but, with injections of ½ drachm of ½ per cent. cocaine to which 4 drops of 1:1000 adrenalin solution have been added, he has been able to do long operations upon deviated septa. In hypertrophied turbinates, where the ischæmia produced by cocaine increases the difficulty of operation, the author prefers as an anæsthetic a 1 per cent. solution of yohimbine, which does not cause vasoconstriction. Anæsthesine, in the author's opinion, lessens the dysphagia attendant upon syphilitic and tuberculous conditions of the glottis.—*Berliner klinische Wochenschrift*, 1905, No. 8, p. 211.

Formic Acid and its Salts.—Dr. H. Huchard considers these substances vigorous muscular tonics and diuretics. Sodium formiate is preferable to the acid and to other salts, and may be given in dosage of 45 to 60 grains per day, mixed with syrup of orange-peel. The author has never used the drug hypodermically. In asthenic conditions, such as are seen in convalescence from the infectious diseases, in patients affected with cardiac and arterial lesions, etc., the muscular system becomes much more vigorous and the body capable of much more exertion after the administration of the formiates, as is proven by the use of instruments calculated to measure muscular power. These substances also have a noticeable diuretic action. The urine is increased very shortly after the drug has been given and the diuresis ceases a day or two after its suspension. A point especially noteworthy concerning the effect of the medicament is that the output of urea, as well as the quantity of the urine, is markedly augmented. Long usage of the formiates does not seem to establish a tolerance, no toxic manifestations have been noted, nor is there any irritant effect upon the kidneys.—*Revue de Thérapeutique*, 1905, No. 7, p. 217.

Potassium Tellurite in Profuse Sweating.—Dr. Pochorecki has employed this substance in doses of from 0.15 to 0.75 grain daily. In the night-sweats of pulmonary tuberculosis, 0.3 to 0.5 grain, taken in the evening, prevents this unpleasant symptom, and in mild cases smaller doses are effectual. Rarely after long usage does the dose have to be increased, and while in some cases the larger doses may cause intestinal disturbance, smaller dosage is not subject to this disadvantage. The great drawback to this remedy is its alliaceous odor, but, fortunately, this is not perceived by the patient, even though it is noticeable to others, for some weeks after the cessation of its administration. The author has also employed this substance in healthy subjects who are subject to excessive perspiration in hot weather.—*Journal de médecine de Paris*, 1905, No. 15, p. 148.

The External Use of Guaiacol.—Dr. Hecht has used a 10 per cent. ointment of guaiacol and salicylic acid in acute articular rheumatism and in pleurisy with good results. Its employment has caused no unpleasant effects, even in children. In adults the effect of the treatment may not be so quickly apparent as in children, due to the fact that the skin of the former absorbs less easily. In cases of pleurisy with effusion,

many of them probably tuberculosis, the author's results have been uniformly excellent. In early cases he states that the fluid may be absorbed in from five to nine days. If the ointment causes marked irritation of the skin, a new area should be used for each application.—*Munchener medizinische Wochenschrift*, 1905, No. 9, p. 415.

PEDIATRICS.

UNDER THE CHARGE OF

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AND

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Prophylaxis of the Ear in Acute Infectious Diseases.—OSTMANN (*Münchener medizinische Wochenschrift*, 1905, Band lii., No. 15, p. 694) calls attention to the frequency and dangers of middle-ear complications in the course of infectious fevers, to the manner in which they arise, and to the fact that proper prophylaxis will prevent many of them. In some instances the ear affection may be laid at the door of the specific infection, but in most of the cases it is only secondary to this, the infection producing a series of local and general changes, which predispose to the ear disease. In children the Eustachian tube is relatively short and wide, and, therefore, it is easily opened during swallowing, yawning, etc.; the emaciation associated with all the acute infectious diseases removes also a certain amount of the fat of the structures of the Eustachian tube, functionally weakening its tensor muscle and leaving the tube patulous; this favors propagation of the infectious process from the throat.

This possibility of infection becomes almost a certainty in the cases where the nose and throat of the child are already the seat of chronic inflammation or of adenoid vegetations. The already existing organisms are made much more virulent by the infection, and on account of the stopped-up nose, it is comparatively easy for mucus to be pulled into the ear during forced breathing, coughing, etc. Other possibilities of setting up a middle-ear inflammation are through extension of inflammation within the tissues themselves. The nasopharyngeal catarrhs of children, therefore, should be looked after and cured; they lead to accumulation of mucus in nose and throat and prevent the child from breathing properly; the infection makes the condition considerably worse and often makes it impossible to treat it properly. The children should be accustomed at an early date to cleanse their noses and throats through proper breathing and gargling; especially unilateral, vigorous but not violent breathing should be practised. The gargling or rinsing should be performed thus: A small amount of fluid is taken in the mouth, a deep breath taken through the nose, and at the heighth of inspiration the breathing is checked; the mouth is opened, the tongue protruded, and the tongue depressed with depressor; the fluid falls into the pharynx, a choking sensation is produced and the fluid ejected;

after some practice the depressor becomes unnecessary. During the disease it is very necessary to keep the nose and throat clean; if catarrh exists a cocaine and boric solution should be used three to four times daily. The child sitting up in bed leans forward and opens the mouth; the atomizer just within the nose should have its ball compressed gently four to five times, thus cleansing not only the upper but also the posterior part of the nose; the child is then told to blow one side of the nose after the other and large amounts of mucus and pus usually come away. Renewed infection of the throat is prevented, the ear is relieved, and mixed infection is averted.

If the nose is absolutely stopped up, cocaine and adrenalin solution may have to be applied before the spray is used. The teeth and mouth must also be carefully looked after. As ear complications can arise, even after the disease process has been checked, the emaciation and weakness of the child being largely the cause of this, the prophylactic and cleansing measures should be continued for some time longer.

Cases of Sudden or Rapid Death in Pneumonia.—E. TERRIEN (*Revue mensuelle des maladies de l'enfance*, April, 1905, p. 155), referring to the rarity of sudden or rapid death in the course of or convalescence from pneumonias of infancy, quotes cases reported by Parker, Hénoch, Netter, and Viti, in which a purulent pericarditis, alone or associated with other suppurative lesions, was found at autopsy. Leroux has reported a case in a girl, aged five years, in which sudden death occurred on the third day of a pneumonia of the right apex, with hyperpyrexia. The author reports 2 cases of sudden death. In 1, an infant of eighteen months, dying on the fourteenth day, a purulent pericarditis was found, with a small collection of pus in the interlobar fissure.

The second case occurred in a girl, aged ten years, admitted to the hospital for a recent chorea. Ten days after admission the temperature rose in the evening to 101.3°, with vomiting. On the following day the temperature was the same, pulse 100, and respirations 40 to 45; there was dry, frequent cough, and a slight cyanosis of the extremities and ears. The chest showed slight dulness of the right apex, with fine rales over the upper and middle third of the lung and in the axillary region, with blowing breathing. The heart, however, showed no signs. Later in the day the child experienced an attack of suffocation, became markedly cyanotic, and died in a few minutes from asphyxia. The autopsy showed pneumonia of the right upper lobe, but there was no extrapulmonary localization, the right cavities of the heart alone showing great acute dilatation.

The following conclusions are drawn:

1. Extrapulmonary localizations are frequent, especially before the age of two years. Pericarditis, usually suppurative, is frequently encountered. It is often latent and may determine rapid death.

2. Sudden or rapid death may be observed at every age, with or without extrapulmonary localization; it is, fortunately, not a frequent accident. This possibility, in a certain degree, allies pneumonia of the child with other affections, such as diphtheria and typhoid fever, in which sudden death has been observed.

3. Cyanosis is often the first sign which reveals the imminence of a fatal termination, and may be an indication of a pericarditis or of a simple acute dilatation of the heart.

OBSTETRICS.

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The Mortality of Parturition in Hospital and General Practice.—In the *Journal of Obstetrics of the British Empire*, May, 1905, BOXALL publishes the results of his continued observations on the mortality of parturient patients in England, Wales, London, and the Provinces.

His statistics include death in puerperal septic disease, and also what are termed the accidents of parturition; abortion, premature labor, puerperal mania, convulsions, placenta prævia, and hemorrhage. The York Road Hospital furnishes the statistics of hospital practice. This building is an old one, but has repeatedly been altered to secure good ventilation, and for some time antiseptic precautions have been practised within the wards In the last sixteen or more years, among 8373 patients, no deaths from septic infection occurred among those who had been for some time in the hospital, and who had not been examined by outside physicians. During this period 4 deaths took place from septic infection in patients who had been infected before admission. Septic infection from these cases did not spread to other patients in the hospital within the time of antiseptic precautions. The mortality of this hospital formerly ranged from 3.08 per cent. to 1.69 per cent. Just before antiseptic precautions were instituted, 9 patients died out of 63 delivered; as a result the hospital was closed, its ventilation remodelled, and when it was reopened a system of antiseptic precautions was instituted by a committee, of which Lister was chairman.

At present but one douche is given during the puerperal period, and that on the fifth day. Formalin has been substituted for sulphur for fumigation, and the washing of the walls and the furniture with carbolic solution has been discontinued; for a time a modern patented antiseptic salufer was used, but the results were so bad that bichloride of mercury was again employed.

From these observations, the writer concludes that the total death rate from parturition has not diminished in England, Wales, Scotland, or Ireland. In London it has declined considerably. The death rate from the accidents of parturition has declined generally, especially in London, but remains abnormally high in Ireland. For the last ten years the death rate from sepsis has declined in London; in other parts it has increased; the only explanation for this lies in the fact that it is still almost impossible to induce the general practitioner to practice antiseptic precautions in attending cases of labor.

Abdominal Hysterectomy for Severe Concealed Accidental Hemorrhage.—TARGETT (*Journal of Obstetrics of the British Empire*, May, 1905) describes the case of a multipara who went to Guy's Hospital in col-

lapse. It was stated that she had been seized with severe pain in the lower abdomen during the morning and that she had become very faint. The patient had had seven children, and with the last two there had been hemorrhage during pregnancy. During the last gestation hemorrhage occurred during the seventh month and was treated by the use of the tampon. Labor came on, the child being stillborn. The patient thought she was six months advanced in pregnancy.

When she was first seen the os uteri admitted the tips of two fingers; there had been some hemorrhage and the patient was suffering severe pain. The membranes were ruptured and a leg brought down. Some hours later the patient became pulseless; labor did not advance, and a tampon was applied and the patient removed to the hospital.

On admission she was very pale; pulse 140, very compressible. The fundus reached to the ensiform cartilage and the uterus was very tense. The legs were much swollen, and the urine contained albumin. The uterus had evidently enlarged since the patient's admission. The os uteri admitted one finger alongside the leg of the fetus, but the edge of the cervix was very tight and could not be dilated.

In view of the fact that labor had not come on after version, that the uterus had enlarged, that the patient was growing weaker, and that the cervix could not readily be dilated, it was determined to perform abdominal hysterectomy.

On opening the abdomen the uterus was almost black in color. It was brought outside the abdomen and gauze packed about the uterus. When the womb was opened a large quantity of black clotted blood was removed. The placenta was completely detached and was removed with the fetus. Hysterectomy was then performed, the stump being covered with peritoneum. Intravenous saline transfusion was performed and saline fluid left in the peritoneal cavity.

The fetus measured 40 cm. and weighed three pounds. During the first week the pulse fell slowly from 140 to 110, thickening and tenderness were present in the posterior vaginal fornix, and pus was present in the urine. This cleared up and the patient did well until the twentieth day, when she developed thrombosis of the left lower extremity. The right was also involved, but the patient finally made a good recovery.

The reason for declining the vaginal method of operation lay in the condition of the cervix, desperate state of the patient, with the evidently increasing intrauterine hemorrhage. The patient's recovery was ultimately complete.

Intraperitoneal Rupture of the Bladder.—In the *Journal of Obstetrics of the British Empire*, May, 1905, GRIMSDALE reports the case of a primipara, aged twenty-six years. She had a lingering labor under the care of a midwife, who summoned the physician when the pains became stronger. She was delivered by forceps without much difficulty. The patient stated that she had passed no urine for two days before her delivery. The nurse reported that urine had been expelled during the labor pains. The abdomen was very prominent after the patient's delivery and very tympanitic. On the following day the patient was found in collapse. She had vomited coffee-ground material. The abdomen was swollen, and the catheter brought away fluid looking like beef-tea. The patient was at once removed to a hospital and the abdomen opened. The peritoneum bulged through the wound, and when it

was excised about a gallon of blood-stained fluid gushed out. On the posterior surface of the bladder there was an area of discoloration, and near the centre an aperture through which the urine was escaping. This area was excised and the bladder closed with fine silk. The peritoneum was thoroughly flushed out and the abdominal wound closed without drainage; a glass catheter was fixed in the urethra. The patient made a good recovery.

In the literature of the subject, Walsham reports 28 cases treated by suture, of which 11 recovered. Jones has collected 54 cases treated by suture, of which 26 died and 28 recovered. In view of the high mortality the result in this case was very fortunate.

The Treatment of Eclampsia by Parathyroid Extract.—In the *Revue di clinica medica*, March 4, 1905, VASSALE reports his results in the treatment of 3 cases of eclampsia with extract from the parathyroid glands. This extract apparently causes the convulsions to cease. In the case of a child with tetany it was clearly successful.

The Permanent Results of Dilatation with Bossi's Dilator.—LICHTENSTEIN (*Archiv fur Gynäkologie*, 1905, Band lxxv., Heft 1) reports the permanent results of the use of Bossi's dilator in 28 cases. In order to use this instrument successfully, anæsthesia must be profound and the patient must not struggle. The instrument must be very gradually used, and when employed in this manner no tear of the cervix necessarily results. Of the 28 cases, 6 could not be traced after leaving the hospital, 3 wrote that they were in good health, and in 18 cases an examination was made; these comprised weak pains during labor, narrow pelvis, eclampsia, and tetanus of the uterus. Among these 18 there was no tear of the cervix in 11; a very slight tear of the vaginal portion in 2; an extensive laceration, unilateral, and a bilateral tear. In 10 cases the patients were free from any bad after-effects. In 8 there were various complications. It is estimated in cases delivered by various instruments from 25 to 30 per cent. suffer considerable tears.

Incision in the Median Line to Facilitate Labor.—In place of the usual lateral incisions, FLEISCHMAN (*Wiener klin. Wochenschrift*, March 16, 1905) employs an incision in the median line of the perineum in forceps delivery, and in delayed labor from rigid perineum, where it is not necessary to use the forceps. In 60 cases the incision tore further in only 1. In all these cases it healed without complications.

Operation for Appendicitis during Pregnancy.—MAUCLAIRE (*Annales de Gynec.*, April, 1905) reports the case of a woman, aged thirty years, who had appendicitis when four months pregnant. When the abdomen was opened in the middle line turbid fluid escaped. The uterus was congested, but showed no further abnormality to complete the diagnosis. Two other incisions were made, one above the pubes, the other over the appendix. The last revealed fetid fluid, and a gangrenous appendix full of pus was removed after draining through the right lumbar region. A fourth incision was made on the left side, which let out quantities of fetid turbid fluid. Drainage tubes were introduced into the two lateral incisions. The patient recovered, and the pregnancy was not interrupted.

He also reports a second case of chronic appendicitis at the fourth month of gestation. Three months later the patient was delivered of a fetus apparently dead two months.

In discussing this paper, Segond believed that pregnant women suffering from appendicitis should be operated upon at once. In 1 case where pain was the only symptom, he found a large quantity of pus on operation; in another the appendix was gangrenous, the patient in the first month of pregnancy, with no general symptoms.

Surgical Operations during Pregnancy.—FELLNER (*Zentralblatt für Gynäkologie*, 1905, No. 18) reviews quite extensively the literature of obstetrical operations in pregnant patients. He finds that the interruption of pregnancy depends largely upon the physical condition of the mother. Where there is insufficient action of the kidneys the slightest operation is often followed by abortion; on the contrary, operations upon the external genital organs, upon the cervix uteri, the total removal of a cancerous uterus and removal of myomata, and operations for retroversions are seldom followed by interruption of pregnancy. In four-fifths of double ovariotomies pregnancy is not interrupted, even the rupture of the fetal membranes does not invariably produce interruption of pregnancy.

In all conditions where pus forms during pregnancy, whether in the middle ear or in the brain, or attended by phlebitis, or in the lungs or mediastinum, operation is directly indicated. This is true in purulent pericarditis, in phlegmon, suppuration of the gall-bladder, liver abscess, purulent gonococcus pyenephritis, and abscesses in the genital organs. Every effort must be made to prevent labor from occurring soon after the performance of an operation where pus is evacuated. This is especially true in appendicitis, which occurs not infrequently in pregnant patients. The percentage of interruptions of pregnancy in appendicitis is the same in those operated upon and those not subjected to operation. The writer gives this as from 35 to 44 per cent. The prognosis is best in these cases, and the appendix is removed as early as possible. When labor follows soon after removal, prognosis is absolutely bad. In cases where the operator does not remove the appendix, but makes an incision only to evacuate pus, the prognosis is still worse.

Malignant tumors which can be removed by operation and which occur in pregnant patients, should in all cases be extirpated. In diseases of the stomach and intestines for which operation is indicated, pregnancy should first be interrupted, and then the operation should be performed. When pregnant patients continue to do badly with nephritis, with severe lesions of the heart, in twin pregnancy and with affections of the heart and lungs which threaten life, and when retinitis appears, pregnancy should be interrupted. In cases of heart disease complicated by pregnancy, when symptoms of liver complications or failure of compensation occur, pregnancy should be interrupted.

Placental Alterations in Puerperal Eclampsia and Their Significance.—CALORNI (*Annali di ostericia*, 1905, No. 4) contributes a paper upon this subject, giving results of 2 cases of eclampsia, with microscopic examination in each case. He concludes from these investigations that the morbid phenomena of eclampsia are the expression of a most acute toxæmia, which gradually accumulates through the progressive failure

of the organs of elimination. He thinks primitive toxæmia is usually of placental origin, and secondary toxæmia of hepatic and renal derivation. He thinks that eclampsia can occur independently of hepatic or renal lesions. He draws attention to the significance of the internal secretions of the syncytial tissues in these cases. He believes that there does not exist a purely characteristic eclamptic placenta, for we may have eclampsia with a placenta entirely unaltered; in most cases, however, the syncytial zone is evidently altered. He believes that the villi of the chorion carried into the maternal circulation have also some influence in causing eclampsia. He draws attention to the resemblance of chorioepithelioma with the lesions seen in eclampsia.

The Biological Theory of Eclampsia.—In the *Zeitschrift fur Geburtshulfe*, 1905, Band iv., Heft 2, LABHARDT contributes a paper in which he criticises the so-called biological theory of eclampsia advocated by Dienst and others.

From his review of the subject, he concludes that this theory has explained nothing concerning the subject. The placenta and child can in no way be considered as bodies foreign to the mother, and the absorption of their tissues cannot injure the mother's kidneys not other organs.

Cæsarean Section.—From the clinic of Wurzburg DAUBER (*Zeitschrift fur Geburtshulfe*, 1905, Band liv., Heft 2) reports 30 Cæsarean sections in fifteen years' time among 7533 labors, or 1 in 251. The frequency of Cæsarean section is reported by Chrobak at 1 in 548; by Braun as 1 in 402, and by Leopold as 1 in 254. 16 of these cases were celiohysterotomies and 14 were celiohysterectomies; of the latter, 6 were Porro operations and 8 were done by dropping the stump. The frequency of the conservative operation was 1 in 471 labors, and in the radical operations, 1 in 538 labors. 26 of these operations were done for contracted pelvis; 86.6 per cent. of these, 15 were rachitic, and 12 were flat rachitic pelved. One patient had had two living children by induced labor; the others had lost children in difficult labor. There were 10 cases of osteomalacia; there was 1 case of obliquely contracted pelvis; there were 3 cases of uterine myomata, which could not be pushed up out of the pelvis, and 1 case of eclampsia, where other methods of treatment had failed.

Of the 30 cases, 17 were done for the relative indication and 13 for the absolute. In choosing the form.of operation, celiohysterectomy was done when it was believed that the uterus was infected; when tumors were present in the uterus; in osteomalacia; when the patient was suffering from severe disease; and when, after emptying the uterus, postpartum hemorrhage occurred which could not be checked.

In preparing patients for operation, compresses soaked in 2 per cent. formalin were bandaged upon the abdomen for twenty-four hours before operation, if possible. The abdominal surface was cleansed with hot water, soap and brush, alcohol, ether, and corrosive sublimate. The Trendelenburg posture was not employed. The abdominal incision was a large one, and no cases of hernia are reported. The uterus was turned out of the abdomen and the abdominal contents were protected by sterile compresses. In 6 cases the uterus was so large that it was thought best to open it without removing it from the abdomen, and after the removal of the child, to turn the uterus out for suture.

In 1 of these cases the patient had considerable fever after the operation. The transverse incision was employed three times; the anterior incision in other cases; in 3 of these the anterior incision was made at the fundus. The placenta was found under the anterior incision in 6 cases. No effort was made to produce adhesions between the peritoneum and the anterior uterine wall.

The uterus was closed with silk which did not pass through the decidua; two rows of catgut were then employed, one buried in the muscle, the other closing the peritoneum. The first silk sutures were tied over the other two rows. When Porro's operation was performed, a rubber tube was placed about the cervix; the peritoneum was stitched in the usual manner, needles were put through the stump, which was cauterized with Paquelin's cautery and powdered with salicylic acid and tannin. Where the stump was dropped the cervical canal was cleansed with 20 per cent. carbolic acid and peritoneum was stitched over the stump. The abdomen was closed in layers with catgut, and a subcutaneous stitch employed to complete the closure.

Hemorrhage after emptying the uterus was not alarming in any case. Ergotine was given hypodermically before the uterus was emptied.

Regarding the lives of the children, in 2 cases operation was done before the children were viable; of the remaining 28, 25 were born living; the others had perished during labor. So far as operation was concerned, there was fetal mortality.

Among the mothers there were 4 deaths; 1 from eclampsia; 1 from peritonitis, the patient having had fever before the operation; 1 from weakness on the thirty-second day, the patient having osteomalacia; and 1 dying from heart failure, having phthisis and a diseased heart before the operation. In 3 cases, then, operation may have hastened the patient's death, a mortality of 10 per cent. If the case of eclampsia be excluded, the mortality is 8 per cent. Dauber believes from these cases that the indications for the operations should be enlarged in the relative direction.

Six Cases of Cæsarean Section.—In the *American Journal of Obstetrics*, June, 1905, DE LEE reports 6 cases of Cæsarean section in which mothers and children recovered. One of these sections was done because the uterus could be entered only through a fistula caused by sloughing after a previous difficult labor. The pelvis was also contracted. The uterus was removed, and the mother recovered, although she suffered from severe shock.

Other cases were those of contracted pelves, in some of whom the uterus was removed, while in others the womb was allowed to remain. In 1 case, that of a dwarf, one ovary and part of the tube were allowed to remain, although the patient was suffering at the time from gonorrhœa. As might have been expected, the patient had high fever after the operation. One case of osteomalacia was operated upon by the conservative operation, and the ovaries were removed after the uterus had been closed. In 1 patient silk employed as a suture became infected and a sinus tract resulted, which suppurated for eight months. In 1 case the patient did badly after operation until a tight abdominal binder was loosened, when the bowels moved and distention of the abdomen disappeared.

The author's record up to the time of writing has been 10 Cæsarean section, with the recovery of 9 mothers and 9 children.

GYNECOLOGY.

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Hypoplasia of the Uterus.—POLLAK (*Wiener klin. Rundschau; Zentralblatt für Gyn.*, 1905, No. 19) describes under this term the atrophy of the uterus observed in strong, overfed subjects. He regards reduction of adipose as the important point in these cases, through diet, treatment at Marienbad, etc. Intrauterine injections of hot water are highly recommended, several quarts being used at a time a few days before the expected date of menstruation. Lutein tablets are also commended.

[Since the uteri in these cases are atrophied and the os stenosed, it is difficult to see how these injections can be given easily or safely.—H. C. C.]

Pseudoascitis in Connection with Ovarian Tumors.—HOERMANN (*Münchener med. Wochenschrift*, March 21, 1905) calls attention to the fact that the free fluid, large quantities of which are often found in connection with ovarian tumors, is not a true transudate, but is really a secretion from their epithelial lining. The writer cites a case in which he operated for supposed malignant disease of the peritoneum and found that the free fluid in the cavity came from a small opening in a cystoadenoma of the ovary. Although this perforation must have existed for at least a year, symptoms of peritonitis had first appeared only four weeks before. A chemical examination of the fluid showed that its composition was identical with that contained in the cyst. In another case of ascites the opening has closed spontaneously.

The writer advises an explorative section in all cases of ovarian tumor complicated with ascites, since it is often found that the supposed malignant neoplasm is benign.

Traumatism as an Etiological Factor in Malignant Disease.—MAFFEI (*Revue prat. d'obstétrique et de gynécologie*, 1905, No. 5) concludes a study of this subject, based on several thousand cases reported by different observers, with the statement that it is exceedingly difficult to establish in any case the fact that the development of a malignant neoplasm was directly due to local lesion. He admits that this may occur, but thinks that it must be comparatively rare.

Before the surgeon decides as to the direct relation between an injury and the appearance of a tumor at this site, he should consider the questions of heredity, the condition of the part before the trauma, its exact location, character and extent, the time of appearance of the growth, and the absence of any nodule in another place which might be the original focus of disease.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

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The Question of Iridectomy in Glaucoma Simplex.—CHENEY, Boston (*Ophthalmology*, April, 1905) calls attention to the differing opinions in regard to the value of iridectomy in glaucoma simplex. Theoretically, he is in favor of it. He thinks that the question of the tension as determined by palpation is of no great importance in deciding for or against operation. The fact that a glaucomatous cup has developed is for him sufficient proof that the tension is above normal for that eye, be it supranormal, normal, or even subnormal, as compared with what we regard as normal tension. The disk cannot resist the existing intraocular pressure, whether this yielding be due to congenital or acquired want of resistance. Iridectomy in lowering the tension offers the best chance of checking the advance of the disease. Two facts deserve special consideration; one is that the eye may retain useful vision for years if not interfered with, and the other that blindness may quickly follow as a direct result of iridectomy. The patient's age may be the deciding point in many cases. As loss of sight is inevitable without operation in time, iridectomy may be advisable in comparatively young subjects, forty years of age, for example, while in persons of advanced years, the possibility of escaping blindness by death should be borne in mind. In some individuals life will become unendurable, when the prognosis of glaucoma becomes known to them. In such it may be desirable to operate, if only for its moral effect.

Treatment of Infection after Cataract Extraction.—HANSELL, Philadelphia (*Annals of Ophthalmology*, April, 1905), very justly observes that infection after cataract extraction, in spite of all known methods of asepsis, has not been altogether overcome. Statistics from operators of large experience still show that this accident occurs in from 1 to 4 per cent.; Graefe himself had reduced it to 5 per cent. years ago before asepsis was heard of.

Preventive measures include removal of conjunctival and lachrymal disease. While bacteriology is not yet able to furnish a basis as to the advisability of operation, the presence of the staphylococcus, streptococcus, gonococcus, or the diphtheria germ are positive contraindications. The pneumococcus, the diplococcus, and xerosis bacillus do not necessarily lead to purulent disease.

Endogenous sources of infection are to be removed as far as possible; these include cystitis, endometritis, kidney disease, diabetes, phthisis, rheumatism, necrosed bones, carious teeth. Skin diseases, especially

of the face, should have vigorous cleansing methods applied before operating.

Operations cannot always be done upon selected patients. Many operations are forced upon the surgeon—*e. g.*, patients suffering from chronic nephritis or diabetes, etc.

White's plan of preparing the patient for operation has shown its efficacy in the hands of various operators. In more than 500 extractions done at the Richmond Infirmary, there was only 1 case of infection, and that was mild. The method consists in the bath and purgative; the face is washed with 1:1000 bichloride solution, and finally with alcohol. The conjunctival sac is thoroughly irrigated with 1:10,000 and filled with bichloride vaselin (mercury bichloride, 1 gr.; sodium chloride, 5 gr.; boiled vaselin, 6 oz.). The free use of the ointment is claimed to be an effective protection against infection from without.

The preparation of the instrument and their maintenance in an aseptic condition during the entire time of the operation is, of course, a matter of supreme importance.

The signs of beginning infection resemble those of spongy iritis, from which it must be differentiated. In iritis the upper section of the cornea becomes hazy, the anterior chamber shallow, the iris swollen and hyperæmic, and the anterior chamber is partly filled with a dark mass of spongy exudation. Infection appears as a white point in the line of incision, rapidly spreading until the whole cut is a dense white line sharply outlined against the comparatively clear cornea. The anterior chamber is at first clear and later occupied by a dull white mass suspended in its upper section or protruding through the pupil. The wound may be closed or open, usually the former, because the devitalized condition of the eye and the direct cause of the infection acting in conjunction prevent closure. The edges of the incision may unite before the septic process is fairly under way, when the anterior chamber is reformed and maintains its normal depth, at least for some time.

After infection has occurred, while the outlook is bad, it is not hopeless. Local treatment consists in cauterization of the wound, subconjunctival injections and germicides inserted or injected into the anterior chamber. The cauterization is accomplished by means of the galvanocautery, silver nitrate, mercuric bichloride, carbolic acid, and the thermocautery. Darier declares that the last should not be used. He is the special advocate of subjunctival injections, the value of which, however, still remains to be determined. The insertion of iodoform in rods or disks containing up to 70 per cent. of the remedy are unquestionably of service if introduced early enough, the earlier the better. The injection of other substances than iodoform has received little attention, though bichloride, 1:1000, has given good results. Hansell reports a strikingly satisfactory case where this method was employed with the saving of useful vision.

In all cases the general treatment should be stimulating and supporting.

Peritomy for Diffuse Corneitis and Other Affections of the Cornea.—SNELL, Sheffield (*Ophthalmology*, April, 1905), from experience in over 200 cases, advocates this operation in certain diseases of the cornea; it consists in removing a band of conjunctiva, 2 to 3 mm. in width, all around the cornea; in a few cases he has practised a partial peritomy

opposite a particular portion of the cornea, which it was specially desired to affect, but the complete operation is to be preferred.

The operation may be employed as a routine treatment in diffuse corneitis, and may be expected often to materially shorten the duration of this disease with more complete recovery of transparency. It may also be used advantageously in a variety of other corneal affections, especially recurring ulcers of the cornea. The operation appears to be free from danger.

Full Correction of Myopia.—Bourgeois, Reims (*Arch. d'Ophthal.*, May, 1905), in a paper read before the Société Française d'Ophthalmologie, advocates full correction of low and moderate degrees of myopia, especially in young subjects. These form about two-thirds of the total number of myopes. For the higher grades of myopia above 12 D. there is no general rule, each case to be decided by itself. In some young subjects partial corrections gradually increased until the full is reached will often be successful.

Until about twenty years ago oculists had very generally recommended partial correction of all cases of myopia above low degrees. Since then the subject has been largely worked over by oculists of all countries. One of the most recent publications is that of Sattler, in 1904, reviewing the work of most previous writers, and recommending full correction.

Those who have practised full correction have established the fact that this method exerts a beneficial influence upon the further progress of the myopia, arresting or retarding its increase. It is accordingly especially important to prescribe such correction at the beginning, when the error is still low. This is no doubt true of the myopia caused by prolonged near work; but it is not yet proved that it also holds good in malignant or dangerous myopia; this must be treated as regards correcting glasses with great prudence.

There are two methods: full correction at once, best applied in young subjects whose myopia is of low degree, and progressive increase in the strength of the correcting lens in the higher degrees.

The writer holds that cases of myopia up to 1 D. in young subjects will do well with the full correction; when thus early discovered and corrected these cases never increase. Even up to 3 D., the full correction is well borne in young subjects. For the general application of such correction a careful examination of the eyes of all school children must be made by competent oculists. The indiscriminate sale of glasses should be regulated by law, since children who are provided with glasses for their myopia without competent examination almost always select an overcorrection, especially in low degrees of myopia.

The writer rightly holds that skiascopy is the most exact and rapid method of determining the refraction in myopia; but he is wrong, as we believe in thinking that this can be safely done without a mydriatic. If practised in this way the patient's accommodation will lead to overestimation of the refraction in many cases in young subjects. Astigmatism is also, of course, to be carefully considered in the prescription. Anisometropia must receive proper attention; the accurate adjustment of the lenses before the eyes is no less important. Where insufficiency of convergence is present, it may be compensated by decentration of the glasses.

DERMATOLOGY.

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Postexanthematic Lupus Vulgaris.—VON VEIESS (*Monatshefte für Praktische Dermatologie*, Band xl., No. 11), reporting a case of disseminate lupus vulgaris, observed in Unna's clinic, following varicella, declares his belief in the external inoculation theory of the origin of these cases of lupus following the exanthemata. The many lesions of the skin; the absence of metastases in internal organs; the absence of generalized emboli; the appearance of disseminate lupus after and not during the acute exanthem; the frequent increase in the eruption at a later period, all are in favor of the external origin through inoculation. The multiplicity of the lesions, the only argument in favor of the hæmatogenous origin of the affection, is equally in favor of the origin by external inoculation, since the latter may readily occur simultaneously in many places through the numerous traumata existing in the delicate skins of children.

Implication of the vessels in the structure of the lupus nodules, and the appearance of nodules in all the layers of the skin, histological features necessary to the support of the hæmatogenous theory, are wanting.

Moniliform Hairs.—BERING (*Archiv fur Dermatologie und Syphilis*, Band lxxv., Heft 1) reports a marked case of moniliform hairs occurring in a boy, aged five years. The affection began soon after birth, and was accompanied by a well-defined hyperkeratosis of the follicles, not only on the scalp, but on the shoulders and extensor surfaces of the extremities. There was a distinct history of hereditary, the father, a paternal aunt, and a cousin suffering from the same disease. Microscopic examination of a portion of the diseased scalp showed the corium very firm and poor in elastic fibres. Around the vessels were clear evidences of a chronic inflammation, and here and there the glands, more particularly the sebaceous glands, were surrounded by a dense infiltrate. The sebaceous glands were few in number, dilated, and atrophic, their excretory ducts being filled with masses of cornified epithelium. In many cases the ducts were so completely closed as to entirely prevent the exit of the hair. In consequence of this obstruction to the normal growth of the hair, it was greatly twisted and distorted; and the author believes that the spindle-like enlargements upon the hairs result from mechanical causes rather than from any pathological process.

Paget's Disease of the Nipple.—Tschlenoff (*Dermatologische Zeitschrift*, Band xii., Heft 3, 4, and 5) believes Paget's disease must be reckoned among the cancerous affections of the skin, representing an independent form of the same. Clinically, it represents a peculiar affection of the skin which must be distinguished from eczema and other similar diseases, as well as from ordinary cancerous affections of the skin. It occurs not only in women, but in men, and attacks not only the mammary gland, but other parts of the body. Its pathological anatomy presents all the phenomena of appearances of cancer, the chief peculiarity being the presence of the peculiar cells known as Paget's cells, which are seldom seen in other affections of the skin. Those superficial diseases of the skin which precede or accompany cancerous affections are of great importance in the etiology of this malady. Pathologically, Paget's disease is a cancer from the very beginning of its development, and its division into a cancerous and an eczematous stage is of no practical advantage. The large pale cells are not, as was earlier supposed, psorospermia, but epithelial cells, which have undergone a peculiar transformation, the nature of which is not yet well understood. The parasitic origin of this malady seems probable, but remains for the future to decide. The author regards the treatment as entirely surgical.

Effect of the X-rays upon the Normal Epidermis and upon Epitheliomatous Tissues.—Dalous and Lasserre (*Annales de dermatologie et de syphiligraphie*, 1905, No. 4) give the results of recent histological studies in the following conclusions: The study of epithelium shows that special lesions follow the action of the *x*-ray, which the authors designate a true radioepithelitis. Analogous modifications exist in neoplastic tissues. Nevertheless, the epithelial cells are not totally destroyed by the action of the *x*-rays, certain of them appearing to be much more sensitive to the action of the rays than others. On the one hand, the cells in sound epithelium composing the generative layer and those immediately above them and the cells corresponding to these in epithelioma are much more sensitive than the prickle cells and those of the corneous layer. The spinocellular epitheliomata are less likely to be affected favorably than those of the basocellular type. These facts find their explanation in the comparative study of the elective changes which the Roentgen rays produce in certain anatomical elements of the normal epithelium.

Treatment of Inveterate Pruritus Ani.—Charles Ball (*British Medical Journal*, January 21, 1905) speaks in favor of surgical treatment by means of curved incisions on each side of the affected area, enclosing the whole ellipse, except in front and behind. The incisions reach down to the sphincter muscles and the flaps raised by dissection with scissors from the muscles. All bleeding must be stopped before the flaps are replaced to ensure against the formation of a hæmatoma. Retained sutures are used with intervals for drainage. These cases are referred to, showing that the operation gave relief at once and that superficial sensation may be restored without the recurrence of itching.

[This operation, it seems to us, is rarely justifiable, and for the reason that cases are seldom met with that cannot be relieved by less heroic methods.—Eds.]

Dermatitis Seborrhoica and its Relations to Alopecia and Other Conditions.—L. D. BULKLEY (*Medical Record*, May 13, 1905) reviews his experience with dermatitis seborrhoica in private and clinical practice and concludes that it is an eruption that should be recognized, since it forms about one-tenth of the cases which come to a dermatologist. Both sexes are affected in about equal proportions, and though observed at all ages from one year to eighty-nine years, but it is mainly a disease of middle life. Combining the statistics of alopecia and dermatitis seborrhoica, it appears that in 557 out of 880 cases, or 63 per cent., the loss of hair was due to the latter condition. Dermatitis seborrhoica in all its forms is regarded as more or less contagious, and that much of modern baldness is caused thereby. Its development is, however, undoubtedly favored by the various elements which lower the vitality and render the skin more susceptible to operation of local agencies. The author describes his method of treatment, which consists mainly in the application of various combinations of resorcin, sulphur, salicylic acid, carbolic acid, formalin, etc. Proper local treatment is essential, but for the best results reconstructive treatment is necessary, that the skin may not furnish such a suitable ground for the growth of the micro-organisms found in it

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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The Transport of Tetanus Toxin to the Spinal Centres through the Nerves.—TIBERTI (*Centralblatt f. Bakt. u. Parasit. Orig.*, 1905, Band xxxviii. p. 625), from a long series of experiments, draws the following conclusions regarding the transport of tetanus toxin to the nerve centres of the spinal cord. The work in the main part corroborates that of Meyer and Ransom. If one makes a subcutaneous injection of tetanus toxin in a susceptible animal, it passes for the most part into the lymph vessels and from there to the blood; in small part it is absorbed by the nerve endings and thence conducted to the nerve centres. After hypodermic injections of tetanus toxin in one leg the toxin is demonstrable with constancy in the nerve trunks of that leg. The transport of the tetanus toxin to the nerve centres does not take place by way of the lymphatics of the nerve, but through the plasma of the nerve fibres. It is, however, necessary that the axis cylinders preserve their normal integrity in order that the nerve fibres may take up the tetanus toxin and conduct it

further to the nerve centres. Along the axis cylinder the course of the toxin is solely in a cellulopetal direction. The tetanus toxin passes onward toward the nerve cells, probably because the latter exert an attraction upon the tetanus toxin with which the nerve fibres are saturated. If the toxin is injected into the muscle it spreads in the surrounding serous fluid, after which it is absorbed by the nerve endings in the muscle. The tetanus toxin inoculated in the calf muscle of a guinea-pig may be demonstrated in the corresponding sciatic nerve one and a half hours after the injection, while it appears much sooner in the blood, namely, after ten minutes. Small doses of toxin injected directly into the nerves call forth severe symptoms of tetanus, whereas, the same quantity injected subcutaneously leads to no tetanic symptoms.

When a nerve trunk is injected with toxin and a muscle innervated by the nerve likewise inoculated, the toxin seems to be hindered in its transport to the nerve centres, and no disturbances occur in that muscle region. Tetanus toxin inoculated directly into the nerves seems to have no way of reaching the nerve centres other than through the nerve fibres; for if the spinal cord is severed at a certain point the portion of the body above this section is exempt from the effects of the toxin. If a nerve trunk is severed, the muscles innervated by this nerve show none of the symptoms of tetanus when a subcutaneous injection of toxin is given. Direct inoculation of the spinal cord shortens the incubation period and gives rise to the characteristic "tetanus dolorosus." Following intravenous injections, and after a more or less prolonged period of incubation, all the muscles of the body are affected at the same time, because the toxin is absorbed synchronously by all nerve endings. In such cases the so-called local tetanus is always absent. This latter form follows subcutaneous injection or inoculation of nerves. Tetanus toxin inoculated into the circulation rapidly passes into the lymph vessels. Tetanus toxin cannot be demonstrated with any certainty in the spinal fluid.

Typhoid Meningitis.—R. I. Cole (*Johns Hopkins Hospital Reports*, 1904, vol. xii. p. 379) presents a study of meningitis as a complication of typhoid fever. Six cases have been seen in recent years at the Johns Hopkins Hospital in which meningitis occurred as a complication of typhoid fever, and in which the typhoid bacillus was isolated from the spinal fluid. From a study of these cases, and from those reported in the literature, he divides typhoid meningitides into three groups: 1. The cases of typhoid fever with meningeal symptoms, but with no demonstrable lesion or relation to the typhoid bacillus. This is the "meningism" of the French writers. 2. Cases where the typhoid bacillus can be demonstrated in the spinal fluid, but where the inflammation is of a serous character. 3. Cases of purulent meningitis due to the typhoid bacillus.

Cole reports no cases of the first group, but considers that it is probable that more careful investigation will show some of the cases to belong more properly to group two. He has collected 8 cases of serous miningitis due to the typhoid bacillus, and adds 5 cases of his own. From a study of this group of cases, the author recommends that in the study of the spinal fluid in suspected cases in the future the following procedures should be adopted: That cultures be made from large amounts, 4 to 6 c.c. of the fluid; that the pressure of the fluid as it flows

from the spinal canal be estimated, and that the cellular elements of the spinal fluid be studied. He considers that in these cases the condition is caused by toxins liberated from typhoid bacilli localized in the spinal canal.

Of the cases of the third group, that of suppurative meningitis, Cole has studied one which he presents in detail, and tabulates with 13 others collected from the literature. The clinical features of this group, as well as the foregoing, are summarized.

The author concludes that the localization of the typhoid bacilli in the meninges bears an important relationship to the cerebral and meningeal symptoms of typhoid fever. Two forms of meningitis, serous and purulent, may be produced by the typhoid bacillus, the former being, according to the author's experience, the more common. His cases well illustrate the great value of lumbar puncture in these cases, both for diagnostic and therapeutic purposes.

The study of the pathological anatomy of the case of purulent meningitis reported by Cole forms a paper by W. G. MacCallum (*Johns Hopkins Hospital Reports*, 1901, vol. xii. p. 411). He found at autopsy the usual lesions of typhoid fever, together with bronchopneumonia, acute cholecystitis, with ulceration and localized peritonitis and acute cerebrospinal meningitis. The spinal meninges were somewhat distended with a yellowish turbid fluid, from which a pure culture of typhoid bacilli was isolated. Similar fluid was found over the cerebral hemispheres.

In the exudate the microscopic study showed a large number of large mononuclear cells, which formed the most striking feature of the lesion. These cells are three to five times the diameter of a polymorphonuclear leukocyte, and are actively phagocytic. Their relative abundance is, perhaps, the only characteristic feature of the lesion, although they were found also in other forms of meningitis. The origin of these cells is discussed at some length, and the author is of the opinion that they arise from cells found in the normal meninges and especially in the walls of the veins, the chromatocytes of Ranvier, or the adventitial cells of Marchand, which Maximow brings into such close relationship with his outwandering cells or polyblasts. Thus, he considers the process almost entirely exudative, which brings the type of meningitis due to the typhoid bacillus into closer analogy with other forms of meningitis than if the chief alterations were considered as due to proliferation of the fixed tissue elements.

MacCallum also discusses the origin of the other cells of the exudate, and describes a lesion of the arteries in which the intima is lifted by collections of cells beneath it so as sometimes to occlude the vessel. This, however, is not peculiar to meningitis due to the typhoid bacillus alone.

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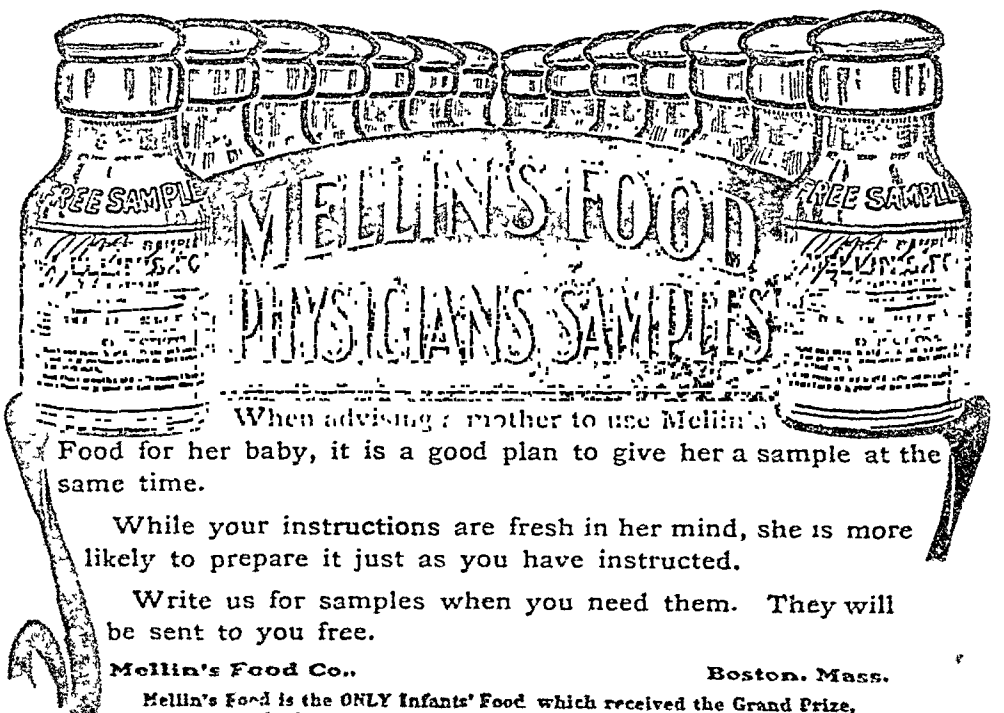


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EDITED BY FRANCIS R. PACKARD, M.D.

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THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES.

SEPTEMBER, 1905.

HÆMATEMESIS FROM GASTRIC ULCER; NOTES ON OVER TWO HUNDRED CASES.[1]

By W. Gilman Thompson, M.D.,
of New York.

The present inquiry was undertaken with the view of formulating the indications for operation in cases of gastric ulcer. The cases have been collected from the records of the Presbyterian and Bellevue Hospitals, and from private sources, numbering collectively more than 200.

During the past sixteen years there have been treated at the Presbyterian Hospital 195 cases in which the diagnosis of gastric ulcer has been made, including 167 females and 28 males—a proportion of 6 to 1.

The most interesting feature of these cases is the fact that so large a number, considerably more than one-half, were discharged as "cured" after serious hæmatemesis which occurred either immediately before admission or during the patient's stay in the hospital, or both. The exact figures are as follows:

Hæmatemesis: "Cured" under medical treatment	111
Improved " "	19
Unimproved " "	3
Died " "	41
Melæna without hæmatemesis	1
Ulcer without hæmatemesis (cured)	20
	195

The total deaths, numbering 41, present a ratio of 1 death to 4.8 cases. Included in the deaths which took place among the unoperated cases were several in which the ulcer was not the whole cause; thus half a dozen patients died from pneumonia, 1 with abscess of the pancreas, 1 with pyonephrosis, 1 with pyopneumothorax, and

[1] Read before the Association of American Physicians, May 16, 1905.

3 with a subphrenic abscess. One patient had a gastroureteral fistula. In 8 of the unoperated cases and in 3 of the 20 which were operated upon, the patients died from the immediate effects of hemorrhage into the stomach, which was found at autopsy filled with blood. In 1 case, for example, the patient lost eighty ounces of blood by actual measurement during the three days that she was in the hospital prior to operation. That such a patient should die upon the operating table is no surprise, although the question might be raised as to the propriety of operating at all, for the situation is very nearly as desperate as operating for a ruptured aneurysm.

Twenty cases were transferred to the surgical division of the hospital for operation, of which 14 proved fatal and 6 were discharged as "cured." Of the 14 fatal cases, 4 presented a septic peritonitis from perforation, and were nearly moribund at the time of operation. Another, as stated above, was moribund from hemorrhage, a sixth was found to have a perforated duodenal (not gastric) ulcer, and a seventh proved fatal on the thirteenth day, a recent perforation having been disclosed at operation. No one of these 7 cases should, therefore, be considered as having any real bearing upon the operative treatment of gastric ulcer, for they all presented a forlorn hope, 2 patients dying on the table and 4 within twenty-four hours.

Among the 7 other deaths one was quite unaccountable. The patient, an Irishwoman, twenty-eight years of age, gave an acute history, including much gastric pain, hæmatemesis and melæna. She had had serious gastric hemorrhage the day before the operation, which revealed nothing abnormal. The stomach appeared normal in size; there was no cicatrix, no visible ulcer, and no hemorrhage. She rallied from the anæsthetic, but died within twenty-four hours with a temperature of 105°. The cause of death, unless it was shock, with unusual temperature, could not be determined. Another patient proved to have a general tuberculous gastritis with hemorrhage. She also died within twenty-four hours. (This case was reported to the Association by F. P. Kinnicutt in 1902.) Another patient died from shock within twenty-four hours after operation. She had been in the hospital a year before for hæmatemesis, from which she was discharged "cured."

It appears that among the 20 operated cases the medical diagnosis was erroneous five times, there being 1 case of tuberculosis of the stomach, 1 of perforating duodenal ulcer, and 3 in which no ulcer was discoverable. From a further analysis of these 195 cases it appears that operative measures can rarely be relied upon for the cure of active hæmatemesis, and that also in many cases operation was delayed until the conditions present were too serious to admit of recovery. When successful results are to be anticipated, they are to be looked for in the category of cases with adhesions, cicatrices, and stenosis chiefly, and in operation such as gastro-

enterostomy performed between, rather than during, attacks of hæmatemesis. In 2 private cases the only operation consisted in separating adhesions which bound the base of an ulcer near the pylorus to adjacent structures (the pancreas, intestine, etc.), and the stomach was not opened. Both patients have remained in the best of health, one for five and one for four years. One is a woman forty-eight years of age who, four years prior to operation, nearly lost her life through excessive hæmatemesis, and suffered constantly thereafter from gastric pain and distress, with almost constant nausea and frequent vomiting. She refused my entreaties to submit to operation until she was so feeble as to make it a last hope, and she so nearly died upon the table that gastroenterostomy was not performed. The adhesions covered the base of an enormous ulcer, 5 by 3 inches across, and bound the stomach to the diaphragm and duodenum. For several days after operation violent hæmatemesis occurred, but none has taken place since, and, as stated above, the patient at present, after five years, is in robust health, eating any sort of food in reason. Prior to operation, for a long time I was able to distinguish two varieties of pain, one apparently referable to the ulcer, and another which appeared to be due to adhesions, and was excited by peristalsis induced by natural movement of the bowels and catharsis, by forced diaphragmatic movements, and by manipulations of the thin abdominal wall, especially by coughing while deep abdominal pressure was made against the diaphragm. The operation proved the correctness of this distinction, one which I have been able to make in several other cases. A third case was that of a woman who had been under my observation for half a dozen years with symptoms of moderate gastric dilatation. She never at any time presented any of the symptoms of gastric ulcer, yet when a gastroenterostomy was performed the cause of the dilatation was found to consist in a pyloric ulcer with cicatrization, which greatly narrowed the orifice. I will cite only two other personal cases in illustration of the difficulties which may attend diagnosis of gastric ulcer, even in the presence of hæmatemesis. In one of these cases the patient was operated upon for persistent hæmatemesis with a typical history of gastric ulcer. A small peptic ulcer $\frac{3}{4}$ inch in diameter was found near the pylorus, bleeding actively from its round periphery; considerable blood was also found in the stomach; the ulcer was excised and the patient made a complete recovery. The other case in contrast was clinically even more complete, yet the operation revealed nothing. The patient, a woman thirty-eight years of age, gave a classical history of gastric pain, epigastric tenderness, distress after eating, repeated attacks of hæmatemesis, with increasing weakness and emaciation, symptoms which lasted through many months. During a fortnight's stay in the ward the patient had serious attacks of hæmatemesis, and as there was no improvement under medical treatment, I advised

operation. The stomach was opened and the surgeon who operated explored the interior thoroughly with an electric light; the mucosa appeared slightly granular and congested, but there was no hemorrhage, no cicatrix, and no sign of any ulcer. The incision was therefore closed, and the patient died three days later from exhaustion following repeated vomiting. Hence, it sometimes happens that diagnosis of the *exact* condition is extremely difficult as between hemorrhage from a circumscribed ulcer which may be cured by operation and that from a generally congested mucosa which may not be so cured. In none of the cases cited in this study has the chemical analysis of gastric contents proved of determining value, for the results have been too variable, not rarely in the same patient on successive days ranging from anacidity to hyperacidity. Additional difficulty is encountered in the variability and inconstancy of all the so-called "cardinal symptoms," and in fact that any one symptom, such as pain, vomiting, or tenderness which has been present steadily for a long time, may spontaneously disappear or give place to another.

I am not yet prepared to accept a definite clinical classification of gastric ulcer into acute and chronic types based upon hæmatemesis, as attempted by Moynihan, Samuel and W. Soltan, and others.

This classification is based chiefly upon the fact that a number of cases occur in which there is a sudden single hemorrhage followed by complete and permanent recovery under medical treatment, whereas, with chronic ulcers there may be repeated small or large hemorrhages and other gastric symptoms recurring at intervals through months or years. Of course, if a patient has a single severe hemorrhage, without protracted previous symptoms, and never has any further symptoms after recovery, the case may be styled "acute," but many chronic cases of which I have records begin in this acute manner. One of the operated personal cases described above was of this type. Again, there may be no hemorrhage and the patient may yet die of perforation.

For these and similar reasons it does not appeal to me to accept a hæmatemetic basis of classification. In other words, a single large hemorrhage may denote a chronic ulcer, equally with an acute one. After all, the important difficulty lies not in trying to separate the gross ulcers into varieties, but in differentiating such cases as the three operated upon at the Presbyterian Hospital in which no gross lesion was found, *i. e.*, the cases of diffuse punctate hemorrhage. These cases suggest those of early hæmoptysis without discoverable bronchial ulceration, and the early hemorrhage of typhoid fever which arises during the congestive stage, before the ulcers are well defined. They also resemble those cases of hæmatemesis which result from congestion in chronic endocarditis and hepatic cirrhosis with portal obstruction, yet no such lesions are found to explain them.

I have not attempted to trace the after-history of the patients who were discharged from the hospital as "cured" cases of hæmatemesis, for owing to the peculiar social conditions existing in New York this is well-nigh impossible. The great majority of hospital cases occur among young women, many of them foreigners, who belong to the social order of domestic servants, seamstresses, factory, operatives, etc. They constitute a floating population to a large extent, and are constantly changing their address, for the servants change their employers, and others rarely occupy the same lodgings for more than one or two consecutive years. If a subsequent attack of hæmatemesis occurs, the patient is quite likely to go to a different hospital, nearer perhaps to a changed residence. It would not be possible to trace successfully the after-history of more than 10 per cent. of this series which extends over a period of sixteen years, and partial statistics of this sort are decidedly misleading.

A better result is obtained, I think, by reviewing the past records derived from the patients on admission to the hospital, many of whom give a history of repeated attacks extending over a period from one or two, to a half dozen years or more, showing that a complete or permanent cure is comparatively rare. Among the cases discharged as "cured" were some in which repeated hæmatemesis had occured during five to six years

To estimate the mortality of gastric ulcer by statistical methods is liable to misinterpretation. For example, J. W. Russell[1] states that 42.6 per cent. of patients recover.

To say that more than 55 per cent. of patients with hæmatemesis received into the Presbyterian Hospital (111 cases out of 195) were discharged as "cured" is of little prognostic significance, for they might nearly all die within from one to six years of hemorrhage, perforation or other complications, and I do not think any patient should be regarded as "cured" unless an interval of at least five years of health shall have followed the attack, and even in such cases the diagnosis may have been wrong, and the original hemorrhage due to congestion, punctate erosions, chlorotic anæmia or other cause, and not to peptic ulcer.

It is admitted that the cases studied for this review do not appear perhaps to make a very strong argument in favor of operation, but many came under operation too late, and most were operated upon before the recent advances in the technique of gastric surgery.

On the other hand, a total fatality of 41 cases among 195 and a possible much larger mortality among those which have not yet run their course, should constitute a plea for earlier surgical intervention in fairly typical cases.

The mortality from cases of gastroenterostomy for gastric ulcer appears to be gradually lessening under improved technique.

[1] *Lancet*, January 30, 1904.

Thus, in 1897 von Mikulicz[1] gave it as 16.1 per cent. for 238 collected cases, and in 1900 Mayo-Robson[2] gave practically the same figure, 16.4 per cent. for 188 collected cases, for all complications except active hemorrhage and perforation, and a year later the mortality rate for his own cases was only 5 per cent. B. G. A. Moynihan's[3] figures are 153 gastroenterostomies for ulcer with only 2 deaths, and in 22 cases of gastric hemorrhage which apparently would have soon proved fatal there were 19 recoveries after operation.

Joseph A. Blake, in an article upon "The Surgical Treatment of Gastric Ulcer," published December, 1904, in THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, states his opinion "that a single large hemorrhage, without previous symptoms referable to ulcer, should not be operated upon, but when there have been antecedent symptoms operation should be performed." Also that "cases suffering from a recurrent hemorrhage should be operated upon."

In these views I concur and would add that those chronic cases also should be operated upon in which the patient's life is either made miserable through pain or endangered through persistent vomiting and emaciation, provided medical treatment tried for two or three weeks has failed to produce any improvement. Let the surgeon be given the advantage of dealing with cases not already made almost hopeless through anæmia, ulcers on the verge of perforation or complicated by a mass of perigastric adhesions. To offset these grave dangers early operation (gastroenterostomy) offers freedom from further hemorrhage, from chronic hyperacidity, pain, traumatic irritation of the ulcer by food, motor insufficiency, and gastric dilatation. Blake makes the further assertion that in the early operative cases the patient may be subjected to less shock than in most cases of appendicitis.

Some additional data derived from a series of 107 cases occurring during the past eight years may be of interest as bearing more or less directly upon the subject and the possibilities of operative interference. Although the ages of the patients range from seventeen to sixty-nine years, 51, or nearly one-half of this series, occurred between the twentieth and thirtieth year, and were observed as follows:

Seventeen to twenty years, 10 cases; twenty to thirty years, 51 cases; thirty to forty years, 34 cases; forty to fifty years, 7 cases; fifty to sixty years, 5 cases.

Of the 28 cases among males, more than one-fourth occurred among those whose occupations predispose to metal poisoning. Thus there were 3 coppersmiths, 2 printers, 3 painters, and 1

[1] Centralbl f. Chirurgie, 1902, 1897, p 69
[2] Philadelphia Medical Journal, May 25, 1901, p 1005.
[3] British Medical Journal, April 8, 1905.

pianomaker. All but 8 were more than thirty years of age. Some years ago I lost a patient from foudroyant hæmatemesis, a man forty years of age, who for three years had taken large quantities of arsenic. A physician had prescribed it for chronic acne and eczema, and the patient, unaware of its baneful effect, had continued its daily use upon his own responsibility. The hemorrhage was the only symptom of ulcer, but it proved fatal during one night and I attributed it to a probable ulcer from this form of metal poisoning.

To the above list of 195 cases may be added 6 which occurred in Bellevue Hospital during the past year, of which 3 were males and 3 females. Of these 1 was cured, 1 improved, 1 died of hemorrhage, and 3 were unimproved—All had hæmatemesis. The ages of the three males were respectively forty, forty-one, and fifty-one years. In January of this year two more cases occurred among males.

One patient, thirty-six years of age, was discharged improved after hæmatemesis, and one fifty-four years of age died after operation with perforation of pyloric ulcer. He had no hemorrhage at any time (he was a painter).

(The 8 cases, added to the previous 195, make a total of 203 hospital cases.)

The influence of social status upon the etiology of gastric ulcer is well exemplified in a comparison of the records of Bellevue and the Presbyterian Hospitals for the year 1904. In Bellevue, with 17,765 medical cases treated during that year, there were only 6 cases of gastric ulcer in the entire service, one of the four divisions having none at all—approximately 1 case in 3,000. In the Presbyterian Hospital, with 1218 medical cases treated, there were 15 of gastric ulcer, a proportion of 1 case in 80. In Bellevue, with 7595 surgical cases treated, but one operation was performed for gastric ulcer (which proved to be a complication of carcinoma), against 1925 operated cases in the Presbyterian Hospital, with 4 for gastric ulcer. The majority of gastric ulcers occur among the servant-girl and seamstress class, patients who are more likely to seek private than municipal hospital relief, either from a species of natural selection or because their well-to-do employers are in the habit of contributing to the financial aid of private hospitals; hence they become interested in them and refer their dependents to them. The municipal Bellevue Hospital abounds in cases of alcoholic gastritis, gastric carcinoma, syphilis, arteriosclerosis, privation and starvation, yet these prove to be comparatively infrequent etiological factors. There were 144 cases classed as "gastritis" in Bellevue in 1904. What is there in the dietetic or other habits of life of the serving-woman class that predisposes to this definite lesion of the stomach wall? It can hardly be their frequent diet of tea and toast, nor tight lacing (Rasmussen); yet what is it? I raise the question as an interesting one, but cannot answer it.

I have examined also the records of cases treated at the Cornell Dispensary since its foundation five years ago. There have been in the General Medicine Clinic alone 13,500 cases, with only one of gastric ulcer—a male, a metal polisher, forty-nine years of age. He had had gastric symptoms for eight months, including hæmatemesis. I was greatly surprised by these data, the more so because Dr. William Armstrong, of my staff, has for several years devoted his time to making analyses of stomach contents as an aid to diagnosis. He reports that among 150 cases lately studied he has not once been enabled to make the diagnosis of gastric ulcer.

These data are of interest in showing the relative frequency of occurrence of gastric ulcer among certain classes of the community.

FINAL NOTE ON CASE II. OF ANGIONEUROTIC ŒDEMA.

By Roger S. Morris, M.D.,
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(From the Clinic of Internal Medicine, Dr Dock.)

In the November number of this Journal for 1904 report was made of two cases of angioneurotic œdema, the second of which was the more important, inasmuch as it was possible to demonstrate a simple, non-inflammatory œdema in a bit of the gastric mucosa, which had been obtained by the stomach tube during lavage after a test breakfast, just at the onset of an attack of nausea, vomiting, and pain. The occurrence of exitus in this case is worth recording, as it resulted from a characteristic, though somewhat infrequent cause.

For the sake of convenience the more salient points in the patient's history may be recalled. The patient, a male, was twenty-one years of age in the fall of 1903, when he was admitted to the University Hospital because of swellings of the hands, legs, and larynx, and "stomach trouble." The mother of the patient has transient swellings of the hands, and one sister suffers from œdema of the face after trauma. Otherwise the family history is negative. The patient had "mumps," "measles," and "chicken-pox" in childhood, with good recovery in each instance. He first noticed a swelling in the hands when he was twelve years old. It usually disappeared within forty-eight hours. Later, swellings appeared in the feet, knees, and elbows, the scrotum and penis. At the age of seventeen the patient had "scarlet fever" with "nephritis," and at the same time he had an attack of "œdema of the glottis," for which tracheotomy was performed. He was unconscious at this time. He was

confined to bed for three weeks after the operation, during which time he says his temperature once reached 106°. It was about two months before the tracheotomy wound healed; previous to its healing there was a discharge, with a "fierce" odor, from the wound. His throat at this time was so sore that he could eat no solid food. After recovering from this illness the patient was in fairly good condition, except for occasional swellings, until April of 1903, when he had another attack of œdema of the larynx. This came on in the morning. At noon he was unable to eat any dinner, and at 6 P.M. of the same day he was unconscious. A second tracheotomy was performed. On July 13, 1903, the patient noticed a swelling "under the shoulder-blade" on rising in the morning. He was aware of swelling in the throat about 4 P.M. After supper he gradually became worse until midnight, when intubation was tried. This failed, and a trocar was thrust into the trachea. The patient was confined to bed five days at this time. About the middle of August he again had symptoms of œdema of the larynx, which subsided with steaming. Dr. Dock examined his larynx October 24, 1903, and dictated the following note: Larynx is small; epiglottis straight, but curled from side to side. Interarytenoid space deep. Entire mucous membrane red and slightly swollen. False vocal cords project inwardly, almost covering the true cords in phonation. Cords slightly red. Trachea small, moderately congested. Neck shows a scar about 3 cm. long, the width varying from ¼ to 1¼ cm.; skin pinkish over it. On swallowing, the lower half of the scar moves upward, being attached to the structures beneath. While in the medical clinic the patient's voice was husky, so that he could not speak above a loud whisper; he had transient swellings in the extremities, and an attack of nausea, pain, and vomiting, alluded to previously.

Since leaving the hospital the patient has been under the care of Dr. Floyd H. Randall, to whom I am indebted for the notes on his subsequent condition. Since the autumn of 1903 the patient has had two attacks of œdema of the larynx, the second of which proved fatal. The first occurred on July 15, 1904. Dr. Randall was called about 10.30 P.M., and found the patient extremely dyspnœic and somewhat cyanotic; he inspired with great difficulty. A hypodermic injection of one-quarter of a grain of morphine was given; he was gradually relieved after about three hours. Because of the patient's distress, examination of the larynx was not possible. The swellings on other parts of the body, especially after blows, were very frequent, more so during the last few months, the patient's father thinks. The fatal attack occurred January 3, 1905. It must have come on very acutely, for the patient was apparently as well as usual when the family retired for the night. About midnight the parents were aroused by an unusual noise, and going to the patient's room found him propped up in bed, the front of

FIG. 1



Photograph of fresh preparation showing larynx and neighboring tissues and organs from Case II. of angioneurotic œdema. Extreme œdema.

FIG. 2



Photograph of fresh preparation showing larynx and neighboring tissues and organs from Case II. of angioneurotic œdema. Extreme œdema.

his night-shirt covered with blood. Dr. Randall was summoned at once, but exitus had already occurred when he arrived.

Before going on to a consideration of the autopsy findings, it may be well to include the additional valuable points in the family history furnished by Dr. Randall. He tells me that another child of the patient's mother "choked to death" very suddenly when about two weeks old. During the past few months, and especially since the death of the patient, the mother has shown more numerous evidences of angioneurotic œdema, from which she has suffered more or less since childhood. She has never had any throat symptoms.

FIG. 3



Photograph of fresh preparation showing larynx and neighboring tissues and organs from Case II. of angioneurotic œdema. Extreme œdema.

The swellings have been noticed on the limbs and face. Of late her neck has been inclined to swell.

Autopsy was performed on the patient some hours after death. Permission was obtained to examine only the respiratory tract. The mouth and pharynx were filled with blood. The sternum was removed; the lungs showed no special change. The walls of the pharynx, the tongue, larynx, trachea, and upper part of the œsophagus were removed *en masse*. The larynx presented a most extreme grade of œdema, which involved the epiglottis, the aryteno-epiglottidean folds, and the false and true vocal cords, the ventricle

being obliterated; in fact, there was the most intense œdema of all the loose laryngeal tissues above the true cords. Below the latter there was no evidence of œdema. The tissue at either side of the arytenoepiglottidean folds was also markedly œdematous. It is an interesting fact that no trace of scar from the tracheotomy wounds can be found in the trachea. The accompanying photographs, taken from the fresh specimen by Dr. C. H. Baker, illustrate beautifully the condition found.

In concluding this brief note I wish to express my thanks to Dr. Dock for his kindness in placing the specimen, which was sent to him, at my disposal for this report; while my indebtedness to Dr. Baker, and especially to Dr. Randall, is obvious.

EXAMINATION OF THE BLOOD IN PULMONARY TUBERCULOSIS, WITH SPECIAL REFERENCE TO PROGNOSIS.

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AND

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Historical.—The blood in tuberculosis has for many years been the subject of investigation by many different observers. Among the first series of clinical examinations of the blood we find cases of tuberculosis recorded, as these earlier reports were based upon methods of examination so different from those now in use and the details of the condition of the patient are so commonly omitted they are of very little service for comparison with the more recent examinations.

In tuberculosis we have such a chronic condition, one which presents so many different phases, types and complications, and which may affect so many organs, that it is not to be wondered at that the results of blood examinations by different observers vary so widely. It is, therefore, only by taking the statistics, classifying the findings in different types of tuberculosis, and studying them in relation to one another that we can hope to derive any information of value for diagnosis and prognosis.

Hæmoglobin. As this was the first component of the blood to be studied quantitatively for clinical purposes we find a large

series of estimations of this element. The methods varied greatly from the spectroscopic to the purely chemical, which renders the results hard to correlate. That the hæmoglobin is constantly reduced in bone tuberculosis has been shown by Vierordt, Lacker, Bierfreund, and Dane. That, on the other hand, this reduction is not constant in pulmonary tuberculosis of various grades is shown by Leichtenstern, Gnezda, Barbacci, and Oppenheimer, who found fairly numerous cases in which the hæmoglobin was above normal. Quinquaud, Wiskeman, Laache, Neubert, Reinert, v. Noorden, Biernacki, Strauss, and Rohnstein, and Stevens found a decrease in hæmoglobin of varying degree in all cases. In those instances in which the cases have been grouped according to Grawitz's system we find the hæmoglobin is given as follows: Group I.: v. Limbeck, Ewing, and Swan, decreased; Appelbaum, decreased or normal; Cabot, increased or normal. Group II.: Cabot and Swan, reduced; v. Limbeck, Appelbaum, and Ewing, normal or approximately so. Group III.: All the writers found it reduced. The majority, therefore, found it reduced in Group I., normal in Group II., and reduced again in Group III. The amount of reduction varied greatly in the different reports. In regard to the high hæmoglobin finding in Group II. we will refer again in considering the red corpuscles.

Erythrocytes. Andral, Sörenson, Pick, v. Noorden, Zappert, Biernacki, Strauss and Rohnstein, and Claude and Zaky found a constant reduction of the red corpuscles in tuberculosis of various stages. Becquerel, Melassez, Laache, Neubert, Reinert, Cabot, and Da Costa found the decrease was not constant, as in some of the cases the count was normal or above normal. The most marked decrease was in a case reported by Melassez, in which the red cells were as low as 980,000. Oppenheimer believes the count is normal in this condition and only reduced by hæmoptysis. Dane, in bone tuberculosis in children, found the reds, as a rule, not reduced. Where the cases are classified we find the following: Group I.: v. Limbeck, Strauer, Grawitz, Halbron, and Swan give a reduction in the red cells; Stevens, Appelbaum, and Ewing believe they are reduced frequently, but may be normal. Group II.: All the observers give a normal or increased count except Swan and Halbron, the former, however, finding it sometimes increased; Halbron found it constantly decreased. In Group III., all the above investigators found the red cells reduced except Swan, who found them generally much reduced, but increased in 3 out of 15 cases. The evidence in regard to the red corpuscles points to the fact that in cases of infiltration or cavity formation with hectic fever, especially the latter, they are decreased in number. That in cases with cavity formation without fever or complications, and occasionally also in the other two groups, we have a normal or even increased count. This peculiar and striking condition was explained by the earlier

writers by concentration of the blood, and this has been confirmed by later research.

Different observers have given profuse expectoration, sweating, and diarrhœa as causes for the production of this concentration. They commonly, however, found cases in which none of the above causes were present. The experiments of Gärtner and Römer have shed new light on the subject and their deductions have been generally accepted. They show that the extracts of the tubercle bacillus produced an alteration in the relations between the blood-vessels and the tissues in the interchange of lymph, with an excess in favor of the tissues. The findings of Oliver may also have some bearing upon this phenomenon.

The most striking feature that we find in all the reports, in connection with the erythrocytes, is the almost constant disproportion between the red cells and the hæmoglobin. All the observers mentioned above found a greater diminution of the hæmoglobin except Oppenheimer and Biernacki, and in addition to those mentioned Hayem, Huard, Cornil, Hanot, Trousseau, Gilbert, and Labbé have specially referred to this very constant finding. Sörenson was the first to point out the striking similarity of the blood in tuberculosis and chlorosis and Labbé has recently considered the subject in detail.

In regard to the prognosis based on the blood findings one must be very cautious, as numerous observers have found the erythrocytes and hæmoglobin increase in cases which were developing general tuberculosis and advancing toward a fatal termination.

The literature is very meagre upon the microscopic changes in the red corpuscles. Anisocytosis, poikilocytosis, and nucleated reds are rarely found, the last being very infrequent even after hemorrhage. Maragliano believes the presence of polychromatophilia has an unfavorable prognostic significance even in cases otherwise apparently favorable.

Leukocytes. The results in numerous observations upon the number of leukocytes are quite uniform, although the line between different groups is not sharply drawn. The earlier observers, Sörenson, Halla, and Reinert found the white cells were generally increased, and in advanced cases were occasionally low. Pick, Rieder, Warthin, v. Jaksch, Galbraith, and numerous others have found the counts in pure tuberculosis without complications were always normal or even low. Among those who have classified their cases we find that Strauer, Appelbaum, Grawitz, and Halbron all found an absence of leukocytosis in Group I. The leukocytosis increases in Groups II. and III., depending upon the degree of mixed infection. The significance of leukocytosis in tuberculosis is best summed up in Ewing's conclusion that "In pulmonary tuberculosis an increase of leukocytes is usually referable to suppurating cavities, advancing pneumonia, severe anæmia, or hæmoptysis."

Varieties of Leukocytes. As the actual number of leukocytes has proven of so little value from a diagnostic standpoint, especially in early cases, an attempt has been made to derive some information from the relative number of the different varieties of cells. We are here confronted with a great many reports differing greatly, not only on account of different groupings of the cases, but also on account of different classifications of the leukocytes. Einhorn and Neubert both found the polymorphonuclears in excess in various stages. The consensus of opinion among the more recent observers is that where we have leukocytosis in tuberculous cases we have a relative increase of the polymorphonuclears. Appelbaum noted an increase of the transitionals accompanying the rise of polymorphonuclears. Ewing and Halbron found in acute and most chronic tuberculosis the large mononuclears in excess. Galbraith found an increase of large mononuclears under rich proteid diet; and Strauss and Rohnstein received the impression that with the progress of the disease the mononuclears showed a tendency to fall.

As the eosinophiles have attracted special attention in tuberculosis we have decided to consider them separately. Cabot does not consider their presence or absence of much significance. All the other observers consider them of some prognostic importance. Zappert found them decreased in febrile cases with an increase subsequent to, or occasionally before, the disappearance of fever. That they were absent or only few in number in severe cases was noted by Holmes, Warthin, Appelbaum, and Swan. On the other hand, that they were present in early cases or reappeared under treatment has been remarked by Claude and Zaky, Appelbaum, and Galbraith. Halbron thought they indicated the resistance of the organism to the infection. Teichmüller considered their appearance in the sputum in large numbers to be a favorable sign.

Experimental tuberculosis, to which one would naturally look for confirmation of the clinical finding, has unfortunately not proven as constant and satisfactory as one could wish. Achard and Loeper found a relative increase of the mononuclears in acute tuberculosis and in those cases associated with serous effusion. They found in animals inoculated with tubercle bacilli a primary leukocytosis which subsided in a few days, being followed by an increase in the mononuclears, the eosinophiles reappearing more slowly. Claude and Zaky found the eosinophiles appeared in the beginning of the infection and disappeared rapidly in acutely advancing forms, but remained for a long time in the circumscribed fibrous types. They noted especially the presence of transitionals and large mononuclears in those cases which supported the infection well.

The injection of tuberculin for diagnostic purposes corresponds in its blood changes to the results of experimental tuberculosis. All the observers, Bischoff, Rieder, Botkin, and Rille, found a transient leukocytosis which was followed by an increase of the

eosinophiles even as high as 10 per cent., as reported by Bischoff. This observer also noted an increase of mononuclears and transitionals directly after the injection.

The morphological changes in the nuclei and protoplasm of the leukocytes have been studied by Holmes and by Arneth. They have both attempted to derive information from the degenerative changes in the white cells upon which to base not only the prognosis but also the diagnosis of tuberculosis. Neusser found his perinuclear granules (artefacts) in cases which tended to improve.

The following is a report of observations on the blood in 39 cases of pulmonary tuberculosis, in the wards of the Henry Phipps Institute, by the writers, who were assisted in the early part of the work by Dr. Nisbet, of Philadelphia.

In tabulating our cases we have used the Grawitz system, which divides the cases into three groups. Group I.: Infiltration, with no signs of cavity. Group II.: Those having cavity formation, with little or no fever (the original classification also states "and no complications" in this group; as we had only one case of laryngitis, No. 2056, and three with slight intestinal symptoms, Nos. 2056, 2388, and 2919, we have included them in this group). Group III.: Cases with cavity formation and a hectic type of temperature. We have further subdivided each group into "favorable cases," "stationary cases" and "fatal cases," under the last heading including two cases which were rather rapidly declining. The stationary cases are also unfavorable, as they have not improved under favorable circumstances.

HÆMOGLOBIN. ORIGINAL COUNTS.

	I.	II.	III.	I.	II.	III.
40–45	0	1	0		2388	
45–50	0	0	0			
50–55	0	0	1			2843
55–60	0	0	4			2660, 106, 2087, 2672
60–65	0	1	1		2957	73
65–70	1	3	1	2827	3101, 2923, 3060	2737
70–75	3	3	0	2683, 2799, 1694	2056, 3005, 2826	
75–80	1	3	2	951	2760, 1975, 2919	2478, 2879
80–85	3	1	0	1950, 2984, 2758	2266	
85–90	0	3	1		3015, 2674, 2676	1554
90–95	0	3	2		2632, 3065, 2764	388, 3054
95–100	0	0	1			3079

I.	Maximum	84	1950	Minimum 66	2827	Average = 74.8
II.	"	90	3065, 2764, 2632	" 40	2388	" = 75.6
III.	"	100	3079	" 50	2843	" = 70.6

The hæmoglobin in Group I. is quite constantly moderately reduced, averaging 74.8 per cent. In Group II. we find a very slight increase in the amount of hæmoglobin, in the general average, to 75.6 per cent.; in this group, however, we find a great variation in the amount, just as we do in Group III. In the latter we find a decrease in the average to 70.6 per cent., which would be more marked if it were not for three fatal cases which had a very high hæmoglobin percentage. It will be noted that the difference in hæmoglobin in the three groups follows very closely the changes in the red corpuscles.

ERYTHROCYTES. ORIGINAL COUNTS IN THIRTY-NINE CASES.

	I.	II.	III.	I.	II.	III.
2,500,000-3,000,000	0	0	1			73
3,000,000-3,500,000	0	1	2		3101	2660, 2843
3,500,000-4,000,000	1	0	5	951		2478, 106, 2087, 2879, 2672
4,000,000-4,500,000	3	4	0	2683, 1950, 2827	3005, 2056, 3060, 2676	
4,500,000-5,000,000	4	9	0	2984, 2758, 2799, 1694	2826, 3015, 2923, 2266, 2632, 1975, 2674, 3065, 2388	
5,000,000-5,500,000	0	3	3		2919, 2957, 2764	1554, 3051, 2737
5,500,000-6,000,000	0	1	2		2760	3079, 388

I.	Maximum	4,930,000	2758	Minimum	3,920,000	951	Average = 4,510,000
II.	"	5,550,000	2760	"	3,250,000	3101	" = 4,630,000
III.	"	5,870,000	3079	"	2,950,000	73	" = 4,297,000

HÆMOGLOBIN OR COLOR INDEX. ORIGINAL COUNTS.

	I.	II.	III.	I.	II.	III.
0.30 to 0.40	0	0	0			
0.40 " 0.50	0	1	0		2388	
0.50 " 0.60	0	0	0			
0.60 " 0.70	0	1	1		2957	2737
0.70 " 0.80	4	6	3	2683, 2827, 2799 1694	1975, 2760, 2826, 2923, 3060, 2919	2843, 2087, 2672
0.80 " 0.90	2	3	6	2984, 2758	3005, 2056, 2266	2660, 1554, 106, 388 3051, 3079
0.90 " 1.00	2	5	2	951, 1950	2632, 3015, 2674, 3065, 2764	2478, 2879
Over 1.00	0	2	1		3101, 2676	73

I.	Maximum	0.95	951	Minimum	0.73	2799	Average 0.825
II.	"	1.05	2676	"	0.42	2388	" 0.825
III.	"	1.01	73	"	0.60	2737	" 0.831

In considering the number of erythrocytes we find that in Group I. the 8 cases ranged between 3,920,000 and 4,930,000 with an average of 4,510,000. The 18 cases in Group II. varied between 3,250,000 and 5,550,000, averaging 4,630,000. In Group III. 13 cases gave from 2,950,000 to 5,870,000, average 4,297,000. We find, therefore, in Group I. a mild anæmia, in Group II. the average is higher, and that in Group III. we have two distinct divisions of cases, those with a distinctly low count mainly made up of "stationary cases," and a group in which the counts are quite high, almost entirely made up of "fatal cases." The average for this group is lower than either of the preceding groups. In only one instance, Case 1694, was a single normoblast noted.

The study of the color index or the relation of the red corpuscles to the hæmoglobin gives very striking figures. In all three groups the color index averages practically the same and this average is distinctly below normal. In Group I. none of the cases reach normal, ranging between 0.73 and 0.95, averaging 0.826. In Groups II. and III. we have a much wider range in the figures, 3 cases going above 1 and 3 falling below 0.70; the average for Groups II. and III. being 0.823 and 0.831 respectively.

LEUKOCYTES. ACTUAL COUNTS.

	I.	II.	III.	I.	II.	III.
6,000 to 7,000	2	0	0	951, 2799		
7,000 " 8,000	1	1	0	1950	3060	
8,000 " 9,000	1	2	2	1694	2632, 2919	2672, 388
9,000 " 10,000	0	3	0		2266, 2388, 2957	
10,000 " 11,000	1	1	3	2984	2676	1554, 3054, 2879
11,000 " 12,000	0	2	0		2760, 1975	
12,000 " 15,000	2	3	2	2758, 2827	3015, 2923, 2674	2478, 3079
15,000 " 20,000	1	6	5	2683	3005, 3101, 2826, 2056, 3065, 2764	106, 2843, 2087, 2737, 73
Over 20,000	0	0	1			2660 (empyema)

I.	Maximum	16,440	2683	Minimum	6,100 2799	Average = 10,285
II.	"	19,200	2056, 2764	"	7,600 3060	" = 12,772
III.	"	20,640	2660	"	8,800 388	" = 14,041

The leukocytes show marked variation in all the groups, the average, however, increasing gradually from Group I. to Group III. We found no typical condition for any one group.

The differential count of the leukocytes in the stained specimen gives information which one would expect to be of value in regard to the diagnosis of this condition. The findings in the present series of cases are rather contradictory and confusing. We found that the actual number of each variety was more significant than

the relative percentage and we have therefore used it in all our considerations.

LYMPHOCYTES. ACTUAL NUMBER IN THIRTY-NINE CASES.

		I.	II.	III.	I.	II.	III.
	300 to 600	1	5	5	2799	3060, 1975, 2760 2923, 2764	2879, 1554, 2672 3054, 3079
	600 " 900	0	4	0		3101, 2826, 2919, 2957	
	900 " 1200	1	4	4	2758	3015, 2266, 3065, 2676	2478, 2660, 2843, 388
Normal	1200 " 1500	3	2	0	2683, 1694, 2827	2632, 2388	
Normal	1500 " 1800	2	2	1	951, 1950	3005, 2056	2737
Normal	1800 " 2100	0	0	0			
Normal	2100 " 2400	1	0	0	2984		
	2400 " 2700	0	1	1		2674	2087
	2700 " 3000	0	0	1			73
	Over 3000	0	0	1			106
	Below 1200	25 %	72.5 %	70 %			
	1200 to 2400	75	22 0	7			
	Over 2400	0	5.5	23			

LYMPHOCYTES. We found in Group I. the lymphocytes were either normal (75 per cent. of cases) or subnormal. In Group II. the majority of cases (72 per cent.) showed a count below normal, as they did also in Group III. (70 per cent.). In Group II. they were increased in 5 per cent., and in Group III. in 23 per cent. of the cases. We have not been able to correlate these rather contradictory findings, except that the majority of the fatal cases were among those showing a lower count.

EOSINOPHILES, POLYMORPHONUCLEARS. ACTUAL NUMBER IN THIRTY-NINE CASES.

		I.	II.	III.	I.	II.	III.
	Below 100	1	9	6	951	3005, 2632, 2266, 3101, 2760, 3015, 2919, 2388, 2676	2478, 1554, 2843, 2672, 2879, 73
Normal	100 to 200	1	6	2	1694	3060, 2826, 2674, 3065, 2957, 2764	3054, 3079
Normal	200 " 300	3	0	2	2984, 2827, 2799		2737, 388
Normal	300 " 400	2	1	3	2683, 1950	1975	2660, 106, 2087
	400 " 500	0	0	0			
	Over 500	1	2	0	2758	2923, 2056	
	Below 100	12.5 %	50 %	46 %			
	100 to 400	75.0	39	54			
	Above 400	12.5	11	0			

POLYMORPHONUCLEAR NEUTROPHILES IN THIRTY-NINE CASES. ACTUAL NUMBERS.

	I.	II.	III.	I.	II.	III.
3,000 to 6,000	5	4	1	951, 1950, 2799, 2984, 1694	2957, 2632, 3060, 2266	388
6,000 " 9,000	1	5	4	2758	2388, 2919, 2676, 2760, 1975	2672, 2879, 3054, 1554
9,000 " 12,000	2	3	4	2827, 2683	3015, 2923, 2674	2737, 3079, 2478, 106
12,000 " 15,000	0	5	3		3101, 3005, 2826, 2056, 3065	73, 2843, 2087
15,000 " 18,000	0	1	1		2764	2660
Below 6,000	62.50%	22.22%	7 69%			
6.000 to 12,000	37.50	44 41	61.53			
12,000 " 18,000	0	33.33	30.76			

TRANSITIONALS. ACTUAL NUMBER IN THIRTY-NINE CASES.

	I.	II.	III.	I.	II.	III.
Below 300	1	0	0	1950		
300 to 600	2	1	0	951, 2758	2266	
600 " 900	1	5	5	2799	2923, 1975, 3060, 2919, 2388	73, 2672, 2879, 3079, 1554
900 " 1200	3	5	2	2683, 2827, 1694	2826, 2632, 3015, 2764, 2957	106, 2087
1200 " 1500	0	6	2		3101, 2760, 3005, 3065, 2674, 2676	3054, 388
1500 " 2100	1	1	3	2984	2056	2478, 2737, 2660
Over 2100	0	0	1			2843
Below 300	12.5%	0	0			
300 to 1200	75 0	61%	54%			
Over 1200	12.5	39	46			

LARGE MONONUCLEARS. THIRTY-NINE CASES ACTUAL NUMBERS.

	I.	II.	III.	I.	II.	III.
Below 300	3	7	2	951, 2984, 1694	2760, 3005, 2632 2674, 3065, 2919, 2676	2672, 388
300 to 600	1	1	9	2799	3015	2478, 2660, 2087, 106, 2879, 73, 2737, 3054, 3079
600 " 900	1	3	1	2827	2923, 3060, 2388	1554
900 " 1200	1	4	1	1950	3101, 2826, 2266, 2764	2843
1200 " 1500	0	1	0		1975	
1500 " 1800	1	2	0	2758	2056, 2957	
1800 " 2100	1	0	0	2683		
Below 300	37.5%	38.5%	15.5%			
300 to 600	12 5	5.5	69.0			
600 " 1200	25.0	38.5	15 5			
Over 1200	25 0	17 5	0			

EOSINOPHILE POLYMORPHONUCLEARS. Here our findings were more definite. In Group I. we found 87.5 per cent. of all cases were normal or above normal. In Group II., 50 per cent. of cases were below normal, while in Group III. the eosinophiles were never above normal. In only 3 cases of all three groups were they found above 500, and they were all favorable.

The estimation of the polymorphonuclears, transitionals, and large mononuclears showed nothing of special significance.

In 2 cases, Nos. 2683 and 73, in which there was a leukocytosis of 16,000, there was a single myelocyte noted.

In 25 of the above cases it was possible to make a second count while the patient was still in the hospital. The second counts were made after an interval of two to two and one-half months. The duplicate counts being made to see if there were any changes in the blood upon which one could base the prognosis. Of these cases 4 were in Group I., 16 in Group II., and 5 in Group III.

Cases remaining under observation. Increase and decrease referring to second count in comparison to first count. Counts made about two to three months apart.

ERYTHROCYTES.

	Increased.	Decrease.	Increase.	Decrease.
I.	051, 2683, 1950	1694	75 %	25 %
II.	3101, 3005, 2826, 2056, 3015, 2923, 2632, 3060, 1975, 2266, 3065, 2919, 2674, 2676, 2764	2760	91	6
III.	2660, 106, 1554, 2843	2737	80	20

The erythrocytes generally increase under treatment; this is true of the favorable and unfavorable alike, the increase occurring in those which are not improving as well as in those which are apparently doing very well. In the first group, however, it was noted that the one case which decreased was the only stationary one in that group and in the third group the only one to decrease was the most unfavorable one in that class. In Group II., however, they almost all increased regardless of the patient's general condition.

HÆMOGLOBIN.

	Increased.	No change.	Decreased.	Increase.	No change.	Decrease.
I.		1694	951, 2683, 1950	0	25 %	75 %
II.		1975, 3005, 2923, 3101, 2919	2760, 2826, 2056, 3015, 2632, 3060, 2266, 2674, 3065, 2676, 2764	0	31	69
III.	2737	2660, 2843, 106	1554	20 %	60	20

The hæmoglobin, as a rule, either remained stationary or decreased regardless of any improvement on the part of the patient. The only case in which the hæmoglobin increased was the most unfavorable one in Group III.

HÆMOGLOBIN INDEX.

	Increased.	No change.	Decreased.	Increase.	No change.	Decrease.
I.	1694		951, 2683, 1950	25 %	0	75 %
II.			3101, 2760, 3005, 2826, 2056, 3015, 2923, 2632, 3060, 1975, 2266, 2674, 3065, 2919, 2676, 2764	0	0	100
III.		2737	2660, 1554, 106, 2843	0	20 %	80

As one would naturally expect in a condition where the red cells increased in number without a corresponding increase in hæmoglobin we found an almost constant decrease in the color index.

LEUKOCYTES.

	Increased.	Decreased.	Increase.	Decrease.
I.	951, 2683, 1950, 1694		100 %	0
II.	3101, 3015, 2923, 2632, 3060, 1975, 2266, 2919	2760, 3005, 2826, 2056, 3065, 2674, 2676, 2764	50	50 %
III.		2660, 1554, 106, 2843, 2737	0	100

The leukocytes present a rather peculiar condition and one which is contrary to the usual findings. In Group I. every case showed an increase of the leukocytes under treatment. One-half the cases in Group II. present an increased count and one-half a decrease, in the latter being the majority of the unfavorable cases, and only one stationary case being found in the first half. In Group III. in every case the leukocytes were found to have decreased.

LYMPHOCYTES COMPARATIVE COUNTS.

	Increased.	Decreased.	Increase.	Decrease.
I.	951, 2683, 1950, 1694		100 %	0
II.	3101, 2826, 2056, 2923, 3060, 1975, 2266, 2674, 3065, 2764	2760, 3005, 3015, 2632, 2919, 2676	62.5	37.5 %
III.	2660, 2843	106, 1554, 2737	40	60

LYMPHOCYTES. We feel that in the actual number of this form of leukocyte we have perhaps one of the best indications upon which to base the prognosis, although it is not absolute. In Group I. we found an increase of 100 per cent., in Group II. only 62.5

per cent. increased, while in Group III. we find only 40 per cent. showed an increase in the number of lymphocytes.

EOSINOPHILE POLYMORPHONUCLEARS.

	Increased.	Decreased.	Increase.	Decrease.
I.	951, 1950, 1694	2683	75 %	25 %
II.	3005, 3015, 2923, 2632, 3060, 2674, 2919, 2764	3101, 2760, 2826, 2056, 1975, 2266, 3065, 2676	50	50
III.	1554	2660, 2843, 106, 2737	20	80

EOSINOPHILES. This variety, in a general way, followed the same course as the foregoing. In Group I. 75 per cent. increased; in Group II., 50 per cent. and in Group III., 20 per cent. of the patients showed an increase of the eosinophiles, the one case being a stationary one.

NEUTROPHILE POLYMORPHONUCLEARS.

	Increased.	Decreased.	Increase.	Decrease.
I.	951, 2683, 1950, 1694		100 %	
II.	3101, 3015, 2923, 2632, 3060, 1975, 2266, 2919, 2676[1]	2760, 3005, 2826, 2056, 2674, 3065, 2764	56	44 %
III.	2843[1]	2660, 106, 1554, 2737	20	80

NEUTROPHILE POLYMORPHONUCLEARS. These follow exactly the increase or decrease in the number of leukocytes except in Cases 2843 and 2676, and in the latter one of these the increase in this variety was less than 100.

LARGE MONONUCLEARS.

	Increased.	Decreased.	Increase.	Decrease.
I.		951, 2683, 1950, 1691	0	100 %
II.	3101, 2760, 3005, 3015, 2923, 2632, 1975, 2266, 3065, 2919, 2676, 2764	2826, 2056, 3060, 2674	75 %	25
III.	1554	2660, 106, 2843, 2737	20	80

LARGE MONONUCLEARS. This variety decreased in all of the first group. In Group II., in 75 per cent. of the patients, they increased regardless of the character of the case, and in Group III. 1 case out of the 5, which was a stationary case, showed an increase.

[1] Only cases in which the polys. did not correspond to increase or decrease of leukocytes, q.v.

TRANSITIONAL FORMS.

	Increased.	Decreased.	Increase.	Decrease.
I.	951, 2683, 1950, 1694,		100 %	0
II.	2923, 3060, 1975	3101, 2760, 3005, 2826, 2056, 3015, 2632, 2266, 2674, 3065, 2919, 2676, 2764	19	81 %
III.		2843, 106, 2660, 1554, 2737	0	100

TRANSITIONALS. These showed a very interesting change. In Group I. they were increased in every case, while in Group III. every case showed a decrease. Group II. showed a decrease in 81 per cent. of cases. All those in this group that showed an increase of this form were improving cases.

From these examinations we would draw the following conclusions, being fully convinced, however, that the series is too limited in number and the field covered too narrow to draw deductions which could be considered by any means final:

1. In pulmonary tuberculosis without cavity formation a mild anæmia, with a decrease in erythrocytes and a relatively greater decrease in hæmoglobin, is constant.

2. From the standpoint of prognosis an increase of the erythrocytes, in cases without cavity formation, is of favorable significance.

3. In advanced cases a decrease of the leukocytes is of unfavorable import.

4. In our investigations we have received the impression that the actual increase of lymphocytes seems to correspond to the increase of resistance on the part of the organism to the tuberculous infection, but further study is required to confirm this deduction.

5. The transitionals seem to follow the same rule as the lymphocytes in this regard.

6. At the beginning of the investigation the eosinophiles seemed to increase with the patient's improvement, but further study did not support this view.

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REST IN THE TREATMENT OF LARYNGEAL AND PULMONARY TUBERCULOSIS.[1]

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It is a well-known fact that it is impossible to repair a watch while it is running. It is equally true that rest, partial or complete, is necessary to restore any diseased limb or impaired function or organ of the body. If the eye should be diseased we must rest that; if a limb should be wounded we must rest that; why, therefore, should we not be equally stringent that a diseased throat should be rested?

By resting the larynx, I mean total cessation of phonation, the amount of cough being limited by judicious local and constitutional treatment to the least possible quantity.

Certainly, next to violent paroxysms of cough, and perhaps exceeding them, reckless and prolonged phonation does more toward destroying the laryngeal mucous membrane than any other factor.

We have all borne witness to this truism in the cases of preachers, lawyers, singers, hucksters and all those whose livelihood depends to a greater or less extent upon vocalization.

Never will I forget the impression made upon me many years ago by a preacher whose vocal integrity had been restored by a few months' rest in a mountain region. On his return home, after describing his trip to numerous friends, preaching, etc., he became entirely aphonic, and he early filled a consumptive's grave.

It is not alone in cases of laryngeal tuberculosis that complete rest of the larynx and pharynx is indicated. In ordinary pulmonary cases efforts to phonate excite hypersecretion and contraction of the laryngeal muscles. Therefore, complete cessation of phonation will lessen the amount of cough and consequent prostration, hyperpyrexia, perspiration, etc.

It has been found that this absence of phonation does not result in ankylosis of the joints, even after a period of several months. The explanation of this fact appears to be that in ordinary respiration there is still sufficient motion to prevent it. It might be well for us to recall here that we are not concerned alone with the larynx, because we know that vocalization can be entirely destroyed by traumatism or other interference with the integrity of the pharynx.

During the respiration or swallowing the laryngeal muscles are in a state of comparative rest, but in coughing or phonation every muscle is active. Hence if the integrity of the mucous membrane of the larynx has been destroyed, rest, partial or complete, is imperative before the restorative processes might naturally be expected to set in.

[1] Read before the American Laryngological Association, June 1, 1905.

It has been urged by many writers that cough up to a certain amount should be encouraged instead of hindered, in order that the bacilli may be ejected. A limited amount of cough may be said to be unavoidable in some cases to remove accumulated excretion, but certainly it should in no wise be encouraged; first, because the patient could derive but small benefit from the ejectment of a certain number of bacilli when the tissues were left saturated with them, and second, because we cannot account for the manner in which nature absorbs or throws off the bacilli in some cases in which they appear to have disappeared.

Some sanitariums north and west have achieved great success by reliance alone upon the open-air treatment, with almost total exclusion of all local and even constitutional medication. It would appear, therefore, that if such good results were to be obtained alone by the aid of atmospheric invigoration that Nature was quite competent to take care of the bacilli; but if this is the truth, how much greater and quicker results should accrue with the aid of complete rest of the diseased organ, combined with the judicious use of anodynes, internally, and local applications to heal intractable ulcers, etc.

It has been and still is advocated by some laryngologists that in order to obtain total and complete rest, the respiratory function should be abolished by the aid of tracheotomy. This would, indeed, be ideal, were it possible to obtain such results by this measure. Unfortunately, however, apart from the infection of the wound by the bacilli, the secretions become agglutinated by the inflammatory exudates from the wound; the cough is aggravated, thereby liability to pneumonitis is set up, and the efforts to get rid of the sputa are rendered so difficult that the advantages gained are far more than offset by the disadvantages.

The great benefit derived from the non-use of the voice is so striking that no one can fail to appreciate it who watches the progress of a case. The simple effort to phonate is aggravated a thousand per cent. when the larynx is whipped up to do its normal amount of work, namely, the overcoming of the noises of travelling, other voices, and the continuous effort to do even more than is ordinarily required of it.

A patient recently under my care reported that he had been coughing over a year. He had been rooting at football games, taking part in college sports, and teaching school, with the poorest hygienic surroundings. Of five physicians who had attended him, none had urged him to discontinue the use of his voice. Severe infiltration of the entire laryngeal mucous membrane was found, but the arytenoid prominences were not enlarged, nor was there any ulceration of the vocal cords or of the epiglottis. There was marked dulness under the left clavicle, loud vesicular murmur, and engorgement at base of the left lung as a result of old pneumonia. Tubercle bacilli

were found in his sputa, and he had a temperature varying from 99° to 101°. On the slightest exertion his pulse ran up to 100 and over.

Complete cessation of phonation was exacted of him; he was given small doses of Dover's powder and quinine at night to secure sleep, and an emulsion of turpentine and ammonia, with morphine sulphide, gr. $\frac{1}{6}$ ter in die. He also took, at intervals, palmiacol, thiacol, beechwood creosote, and locally applications of formalin (10 per cent. solution) to the larynx. Occasional applications of nitrate of silver were also made to the pharynx. He was ordered to drink milk in place of water, and to eat as many raw eggs as he could assimilate.

As a result of this treatment his symptoms have all abated, he has gained flesh steadily, the laryngeal inflammation has almost entirely disappeared, and he is deeply impressed with the benefit derived from the non-use of his voice.

It is interesting to note that the improvement in this case has taken place without any special reference to climate. He has not been sent off to the mountains, nor required to sleep out-of-doors, nor to do anything else, except to keep fresh air in his room and to stay in when the weather was inclement. Had he failed to respond to this rest and conservative treatment, he would undoubtedly have left for some higher clime, as so many persons do who are deluded with the idea that their only chance of cure lies in a high and rigorous climate. Many of these unfortunates sacrifice all the care and attention which they would receive at home for the discomforts and even privations which they often find unavoidable among strange surroundings. No case of tuberculosis should be allowed to leave home unless he can get every luxury usually obtainable only by the wealthy, with the care of a skilled nurse and medical attendant.

The chief advantage which patients obtain at sanitariums which they do not get from their home physicians is that more absolute control is exerted over them. Their daily outgoings and incomings are more closely watched. For example, to exact absolute silence by a home physician would be regarded as unnecessarily strenuous treatment, even in the laryngeal cases, whereas it is no less indicated in the one than in the other.

It is astonishing how many so-called cures have been recorded of laryngeal tuberculosis by different authors and under different forms of treatment where patients were often allowed to follow their daily avocations, use tobacco, smoke cigarettes, etc., and yet no one has denied the efficacy of complete rest to the throat, or that the effect of tobacco was injurious.

Failure to observe the precautionary measure of laryngeal and bodily rest in tuberculosis during any degree of pyrexia is simply criminal. This being the case, is it not equally indicated in the apyretic stage?

By complete rest we increase the physiological power of the patient to resist the disease, and, as there is no drug or serum so far discovered which has proved to be a panacea, we are forced to rely on rest, fresh air, good food, and those drugs which we now know limit all inflammatory processes.

In conclusion, we must estimate the amount of advantage to be derived from rest and complete absence of phonation in tuberculosis of the respiratory organs. The violence and amount of cough determines the amount of injury done to these organs and the consequent amount of pyrexia. Every symptom productive of cough is at variance with a return to health. Rest, either partial or complete, should be paramount in all cases.

THE RELATION BETWEEN ANTHRACOSIS AND PULMONARY TUBERCULOSIS.[1]

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We all have heard that miners have a tendency to immunity from tuberculosis, that coal dust prevents tuberculosis, and that it even has a curative effect when tuberculosis is once developed. The present article is an inquiry as to whether these ideas have any basis in fact, and if so, what is the cause.

An investigation into the relationship between any disease and an occupation or mode of life is always complex. This complexity becomes more marked when the disease in question is as intricately connected with social, sanitary, economic, racial, and other factors as is pulmonary tuberculosis. It is doubly so when the human factor to be considered is as varied as to race, habits of living, diet, intelligence, etc., as are our anthracite mine-workers.

Some general aspects of the question will to some extent account for a part of the presumed low mortality from tuberculosis among miners. We know now for instance that tuberculosis is essentially a disease of those living under crowded conditions both at home and at their work. Crowding at work is never present among mine-workers. Seldom are there more than two or three and often only one man working in a large chamber. Furthermore, the spread of infection among the men at work is practically impos-

[1] Read before the Laennec Medical Society, at Baltimore, May 22, 1905.

sible as the moisture in the mine prevents the sputum deposited from drying. The germs must either die out from lack of nutriment or be washed away by the mine waters.

Conditions above ground also favor a low tuberculous death rate among miners. While in the mining region a far too large number of foreigners will often occupy a small house, there is none of the vast overcrowding of the city tenements. A very large number of miners, at least as compared to many other trades, live with only their own families in separate, single, or double houses. Even the houses overcrowded by foreigners are separate so that the number exposed to infection from a consumptive boarder is comparatively small.

The habits of mine-workers as compared to many other working people also tend in a measure to freedom from tuberculosis. For instance, the mine-worker frequently has to walk several miles to the shaft through the fine air of high altitudes. There is comparatively little travelling to and from work in a sputum-soaked street car. Furthermore, a miner's house being generally on a small lot, his time out of the mines is much more frequently spent out-of-doors than is the case with tenement dwellers. Often some of the time is spent working in a small garden. The fact that a daily full bath is imperative also must have a marked influence on the health of miners.

It is of course a fact that for every trade a large proportion of its tuberculosis mortality belongs really to the saloon. The alcohol factor among miners probably does not differ much from that of other workers of the same social grade. These general factors cannot account for all the difference in mortality from tuberculosis among miners that really seems to exist. It is probable that there are other factors among which anthracosis seems to be important.

Before going farther it will be well to consider what evidence there is that this supposed low mortality among miners is an actual fact. One naturally turns first to statistics. In considering the statistics of a disease like tuberculosis in relation to any occupation it has well been pointed out by Miss Brandt[1] that there are two important factors to be considered as having an influence. One is whether there is for a given occupation a sort of natural selection at work by which only the stronger youths take up the occupation, so that from the start we have a more or less picked class in whom tuberculosis would naturally be rare.

Second, is it a fact that miners who have contracted tuberculosis seek lighter work, and finally, on death, are registered with this second occupation to which they really do not belong, thereby artificially reducing the true death rate among miners and swelling it in another easier trade?

In regard to the first question it does not seem that any one who is at all familiar with coal-mining people will think that the

process of natural selection works in any way to make them a picked class. Boys and men take up mining not because they are particularly strong but because it is the easiest work to obtain in the region in which they live, and it becomes in a way their natural destiny.

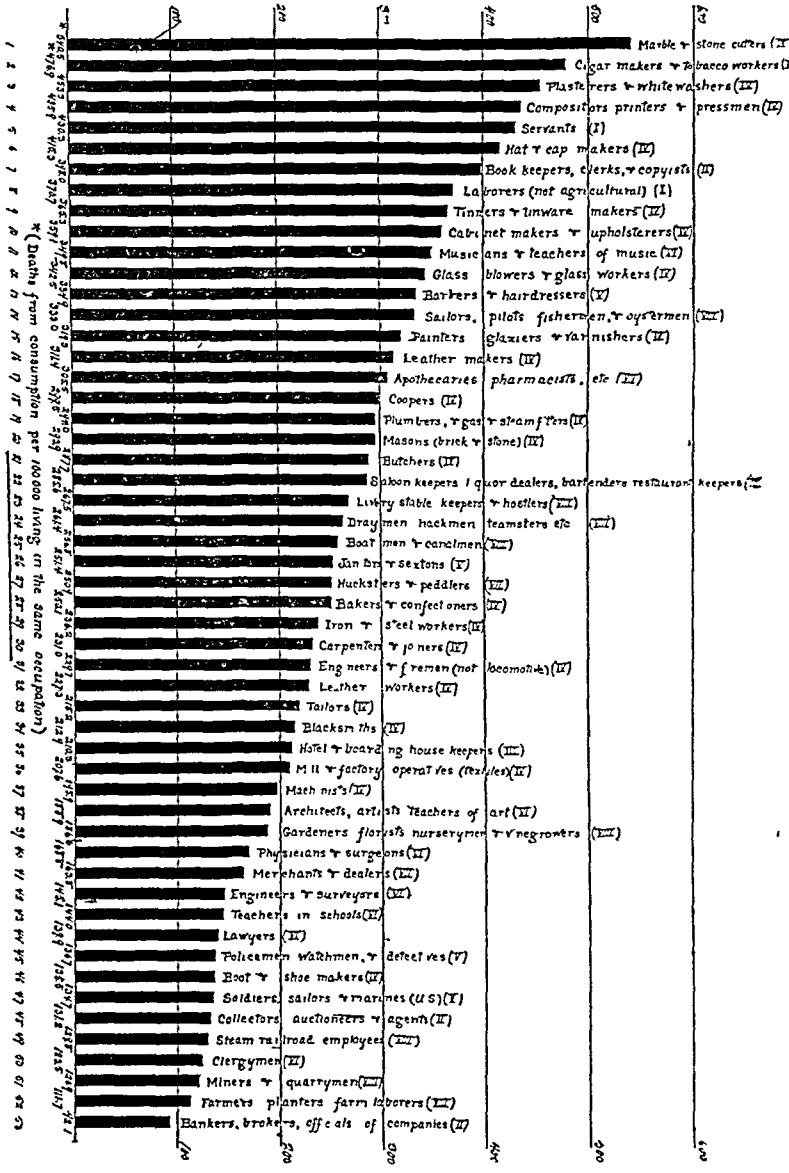
As to whether weaklings even when admitted to the mining occupations are not weeded out later it may be said that mining is not such a laborious trade as to have this effect. It is certainly not more laborious than that of the farm laborer, with whom they are nearly equal in point of tuberculosis mortality. It is certainly not more laborious than that of blacksmithing, where the mortality is about twice as high.

In regard to the second point, whether consumptive miners do not drift into another trade, it seems that here, too, there is not an appreciable factor. Most consumptives continue at their occupation so long that when they do have to give up they do not have enough strength and ambition left to start a new trade. At death they are registered in the trade which they first followed or among the "unemployed." That the "unemployed" contain many who contracted tuberculosis while in active work is unquestioned, but this factor should not balance in all trades.

We think that Schaffer,[2] of Pottsville, Pa., would have agreed with us that these two factors do not operate in the cases of coal miners. He says of the boy starting to work in the breaker that "He grows up to be a lusty, sinewy youth who seldom troubles doctors except from accident." Schaffer quotes Roy, a mine inspector in Ohio, as follows: "With currents sufficient to carry off noxious gases, smoke, and at least most of the dust, mining becomes not merely a healthy but a pleasant occupation, notwithstanding what has been said about its drawbacks . . . men who go from the farm and shops to work for a season in the mines rarely go back to their old work."

The largest collection of statistics bearing on this matter is probably that of the United States Census for 1900. In classifying deaths according to occupations it is unfortunate that the census bureau groups all miners and quarrymen together. On this account it is impossible to get for the whole United States accurate figures concerning coal-miners alone. However, it is to be remembered that all authorities consider colliers the healthiest class of miners, and all agree that stone-dust is much more injurious than coal-dust. It is therefore unquestionably true that the figures for coal-miners alone would be more favorable than those for all miners and quarrymen combined. The following comparison of this class, then, must be read with the knowledge that the comparison for coal-miners alone would be more favorable still. In the census classification from the registration area of fifty-three occupations for the census year 1900, miners and quarrymen occupy the second

place from the bottom in point of view of tuberculosis mortality. The two classes which have a lower mortality rate are: (1) farmers, planters, and farm laborers, and (2) bankers, brokers, and officials



of companies. The rate of death for miners and quarrymen per 100,000 living in the occupation is 120.9. Comparing this with a few of the presumably more healthful occupations we see the rate for lawyers is 139.9, for physicians and surgeons 168.8, for car-

penters and joiners, 231.0; for draymen, hackmen, and teamsters, 261.4; for sailors, pilots, and fishermen, 333.1, etc. The highest in the list are marble-cutters and stone-cutters at 540.5. The rate for out-door laborers, in which miners are grouped, is 147. These statistics are graphically shown in the accompanying diagram, reproduced from the *Handbook on the Prevention of Consumption*, through the courtesy of Miss Lillian Brandt.

According to the same census report consumption caused 10.6 per cent. of all deaths among miners and quarrymen, as against 16.2 per cent. for all occupied males, and as against 10.2 per cent. for the out-door group, among which miners are included.

In the whole United States consumption caused 9.6 per cent. of all deaths among miners and quarrymen as against 14.5 per cent. for all occupied males, and as against 11.2 per cent. for the out-door group of workers. (The figures for miners and quarrymen are based on a return of 5895 total deaths from all causes.) According to this table, therefore, consumption is nearly one-third less frequent among miners and quarrymen than among other occupied males. As noted above, the rate for miners alone would be still less.

In England statistics point equally strongly to a diminished mortality from tuberculosis among miners. Tatham[3] presents a very succinct statement of the case based on figures from the Registrar-General's office for 1890 to 1892. These English statistics show that the relative figures for deaths from phthisis among coal-miners is only 97 as against 109 for all classes of miners (iron, copper, tin, lead, etc.), as against 115 for farm laborers and 185 for all occupied males.

Parry,[4] from English figures on more or less dusty occupations gives the following table of relative mortality in which coal-miners stand lowest not only in regard to consumption but also in regard to all other diseases of the lungs. In both respects they stand lower than all occupied males.

TABLE I.—PARRY (ENGLAND).

Occupations.	Relative mortality figures. Consumption.	Other diseases of lungs.	Total lung diseases.
Coal-miners	126	202	328
Carpenters and joiners	204	133	337
Plumbers, painters, and glaziers	216	185	401
Masons, builders, and bricklayers	252	201	453
Wool manufacturers	257	205	462
Cotton manufacturers	272	271	543
Quarrymen, stone and slate workers	308	274	582
Cutlers	371	389	760
Filemakers	433	350	783
Earthenware makers	473	645	1118
Cornish (tin) miners	690	485	1175
All males, England and Wales	220	182	402

(Sweeps, coal-heavers, and trimmers are given higher figures than coal-miners.

We have not been able to find any large collection of French or German statistics.

In order to get some first-hand information in regard to our own anthracite mine-workers, as a class, we have gone over the death certificates on file at the Scranton Bureau of Health for ten years and have worked out the percentage of deaths from various causes, the average length of life, etc., among miners as compared with other occupied males. The years covered were the last ten, excluding 1902, the year of the long strike of over five months. A short strike of a little over a month in 1900 was ignored. With a general population of something over 100,000, Scranton has had during the above period 857 deaths among miners and 2656 deaths among other occupied males. In each case the actual certificate of the physician furnished data and only complete certificates were used.

Aside from the ever-present possibility of mistaken diagnosis, there are two special circumstances which make our results suggestive rather than mathematically absolute. In the first place, many certificates were excluded as incomplete, and a slight error may have crept in from this. It is fair to assume that this exclusion affects both groups to the same degree. Secondly, the occupation in many certificates is only given as laborers and it was possible that some mine laborers were so designated. On going over the certificates we came to regard this possibility of error as slight because mining is such a distinctive occupation that we found special reference made to it, or the context showed the exact occupation. With these qualifications our statistics are as follows:

TABLE II.—SHOWING PERCENTAGE OF ALL DEATHS WHICH ARE CAUSED BY VARIOUS LUNG DISEASES AND TRAUMA (FOR LAST TEN YEARS AT SCRANTON).

	Pulmonary tuberculosis	Asthma	Pneumonia.	All lung diseases.	Trauma.
Anthracite mine-workers (857 deaths)	3.37 %	7.0 %	10 96 %	23.45 %	45.57 %
All other occcupied males at Scranton (2656 deaths)	9.97 %	1.58 %	13.85 %	27.86 %	14.15 %

Taking the percentage of deaths from all lung diseases among miners as 100 the relation is as follows:

TABLE III.

	All lung diseases.	Pulmonary tuberculosis.	Asthma.	Pneumonia.
Anthracite mine-workers	100	14	29	42
All other occupied males	114	41	7	54

From our own figures in Scranton it would seem that tuberculosis is about two-thirds less frequent among miners than among all other occupied males. By taking our figures of 3.3 per cent. as the proportion of all deaths among miners due to tuberculosis and

comparing it with the census figures for the whole United States we get the following table:

TABLE IV.

	Percentage of all deaths due to consumption.
Anthracite mine-workers	3.3 %
Miners and quarrymen, whole United States	9.6 "
Out-door laborers, " " "	11.2 "
All occupied males " " "	14.5 "

So far as statistics go, we will rest here. It will be of interest to note, however, that the average age at death in anthracite mine-workers from all causes is 42.89 years as against 46.36 years for all other occupied males in the same city. The average age at death from trauma is 34.15 years among the miners and 36.05 among all other occupied males. From causes other than trauma the average age at death is 49.93 for miners as against 48.05 for other occupied males. In other words, if we exclude trauma miners live slightly longer than all the other occupied males in the same city.

Now, we will examine the opinions of writers who have paid special attention to the hygiene of mining or other occupations. As early as 1863 Kubon,[5] of France, says that "coal-dust is unable to cause pulmonary tuberculosis or even to favor the evolution of pre-existing tubercles. It prevents the development of phthisis."

De Crocq,[6] Belgium, in 1862 said that several physicians had noted the rarity of consumption among coal-miners.

In Germany, Arnold[7] says: "It has been noted that among miners tuberculous diseases are very rare. It has even been considered that there is an antagonism between anthracosis and tuberculosis." Arnold disclaims any personal experience in the matter, but considers that if there is any action it is due to the fact that the deposits of dust prevent the spread of the poison. He does not believe that there is a direct action of the dust on the bacteria.

Seltman, quoted by Burger,[8] found tuberculosis a rare disease both in old and young miners. He also announced that coal-dust exerts a favorable influence on a tuberculous process already present. He says that many physicians have treated tuberculosis of the lungs by the inhalation of coal-dust.

Goldman[9] says that tuberculosis is a rare disease among coal-miners. He says it causes only 1 per cent. of deaths among them. This he attributes to an antiseptic action of the dust. Goldman quotes Hirt, who in 4000 cases of pulmonary tuberculosis found that 22 per cent. had been subject to various kinds of dust, 11 per cent. had not been subject to dust, and only 1 per cent. had been subject to coal-dust. Goldman says that tuberculosis is more prevalent among miners' families than among miners themselves. Basing our opinion on a service of over four years in a large miners' hospital, and also on a somewhat close study of the tuberculosis

problem in the largest anthracite centre in the United States (Scranton) we most decidedly agree with Goldman in this statement.

Dirksen,[10] Germany, says that coal-workers have the best health of all dusty occupations. He says that in Upper Silesia only 0.9 per cent. of all internal diseases among coal-miners are due to tuberculosis. He says that many claim a direct immunity from tuberculosis in coal-miners.

In England, Oliver,[11] of Newcastle-upon-Tyne, who is one of the greatest authorities on occupational diseases, draws a sharp line between lesions due to dust and those due to tubercle bacilli. He does not give much attention to the relative action of one on the other. He does say, however, that "ventilation of the British coal-pits has practically abolished pulmonary consumption among our miners."

Trotter,[12] of Beddingtonshire, an English coal-field, says: "As a disease, phthisis is probably more uncommon among coal-miners than among any other class of workers. . . . I am inclined to believe that the comparative rarity of phthisis among coal-miners is probably largely due to the protective influence of coal-dust or soot." He considers this partly a mechanical action due to the coal-dust and soot clinging to and covering the sharp angles of the mineral matter inhaled and "to a lesser degree to an antiseptic and deodorant action." He says that "the death returns from phthisis in a colliery district show a greater proportion of deaths among females than among males." (See Goldman's and our own opinion above.) "Colliery surgeons almost invariably state that phthisis is not so common among miners as in other districts in which they have practised."

We have been trying to add some experimental evidence to the question of the influence of anthracosis on tuberculosis, and as far as we have gone our experiments have been affirmative. Our method has been to keep guinea-pigs about two months in a box in which coal-dust is constantly stirred up. In this way quite a degree of anthracosis has been produced. We have then taken these pigs and injected a suspension of a pure culture of tubercle bacilli directly into the trachea which was exposed by dissection. In this way one can be quite sure that the tubercle bacilli actually reach the lungs. Control animals which had not been subjected to the dust were also injected in the same way with the same syringe full of suspension so that as far as possible the same dose was obtained for all. In the first series when the pigs were killed the control had extensive tuberculosis of the lungs and abdominal viscera. The anthracotic pig had extensive tuberculosis of the abdominal viscera and of the glands around the tracheal injection, but *the lungs were free.* In the second series the result was the same. Two anthracotic pigs were injected and the lungs of both were free. In the third series the two anthracotic pigs had pul-

monary tubercles, and practically the post-mortem finding did not differ from the control of the series. If, therefore, half of the pigs were *protected from pulmonary tuberculosis* by the coal-dust it creates a strong argument. It is not of course to be presumed that every pig would be protected. The animals thus far tested are too few to be completely conclusive, but a great deal of time will be required to get more data. The conclusion of each experimental series takes about four months. Many series have had mishaps and failed, so that many months have been lost. These experiments are being continued.

We have also obtained a similar result by injecting a suspension of coal-dust into the testicles of guinea-pigs, and later injecting the testicles with a suspension of tubercle bacilli. Pigs which had previously been injected with coal-dust lived on an average forty-nine days. The controls lived on an average twenty-five days.

Having gone as far as is now possible in demonstrating that those who have anthracotic lungs are less susceptible to tuberculosis the cause of this phenomena must be considered.

The most frequently mentioned theory is that it is due to the germicidal action of the coal-dust. We have tested the germicidal action of coal-dust by various methods on tubercle and typhoid bacilli, staphylococcus, vibrio, and several unidentified bacteria, and have found that the dust has no germicidal action whatever. It is true that if bacteria are mixed with sterile dust and allowed to remain in contact with it in a test-tube for several days and then plated a slight inhibitory influence will be observed, but even this prolonged close contact will not kill bacteria. There are also several *a priori* reasons against this germicidal action of the dust.

Dr. Nichols considers that it is possible that the soluble calcium salts which form a large proportion of the ash of coal-dust may at least play a part in the protective influence. He is now working on his theory.

Another idea has been that the rarity of tuberculosis among miners is not due to any protective influence but depends on the fact that the air of mines is comparatively free from bacteria. We have shown by tests that in the air of the chambers this is the case. The air supplied to chambers passes slowly through damp gangways sometimes a half-mile. During this passage many bacteria fall to the ground so that the air when supplied to the chambers has undergone a purification by sedimentation just as takes place in rivers. Our tests were by the ordinary Petri-dish method. The dishes being exposed at the beginning of the intake or air tunnel and again for the same length of time in various chambers. The following are two characteristic examples of results:

TABLE V.—SHOWING THE PURIFICATION OF AIR BY PASSAGE THROUGH INTAKE.

	Number of colonies on dish in three minutes	
	Diamond mine.	Dodge mine.
Beginning of intake	880	390
Average in chambers	54	18
End of return tunnel	15	

That this bacterial purification of the air in which the miners work, together with the fact noted above that tuberculous sputum cannot dry, must have some effect in diminishing tuberculosis cannot well be doubted.

As we have said, however, it is probably not the sole factor. Our own opinion is that the coal-dust in the lungs does exert a real protective influence against the tubercle bacilli, and that it is due to the stimulating effect of the coal-dust on connective-tissue growth. This connective-tissue growth is of course the way in which nature overcomes the tubercle bacilli and anything which stimulates it will be a valuable aid in preventing the bacilli from getting a foothold or in overcoming them when once established.

That the coal dust causes this stimulation of connective-tissue growth is well known. Microscopically, anthracosis may be divided into three stages.

The first is seen in those who have worked in the mines only a few months. In these some of the epithelial cells lining the alveolæ can be seen to be swollen and to contain coal-dust. Sometimes a few larger desquamated cells containing much pigment can be found in the alveolæ, lying loose with some detritus and free dust particles. Even in the early stage dust particles can be seen in the walls of the air vesicles and around the bronchi. In this stage there is no thickening.

The second stage occurs in those who have worked in the mines for several years. In these cases the swollen epithelial cells containing dust particles are much more numerous. The alveolæ contain more detritus consisting of swollen, dust-bearing, epithelial cells, and free dust, and some leukocytes. The pigment in the walls of the air vesicles is more marked. It collects particularly in the connective-tissue septa between lobules, where it is in such large masses as to be easily seen by the naked eye in microscopic sections. These are especially well seen in the septa that run in from the pleura and one will frequently see considerable-sized black triangles with their bases on the pleura showing where some septum has been completely filled up with dust. With this deposit there goes an increase in the connective tissue of the alveolar septa wherever there is much dust. The interlobular septa thicken as does also the peribronchial connective tissue, as here also the dust tends especially to collect. With this stage the bronchial and mediastinal

lymph glands are enlarged and perfectly black. The glands in the lesser omentum are also frequently black from dust swallowed in the saliva. This increase of connective tissue is almost never sufficient to give signs of consolidation. With this stage sometimes comes the condition misnamed "miner's asthma." This is really a chronic bronchitis and emphysema both in symptoms and physical signs.

A third interesting picture is seen in those who have formely been miners for many years but who have not been in the mines for some years before death. In these the signs of irritated swollen epithelium have subsided. The epithelium is again normal and neither the cells nor the alveolæ contain dust. The deposits of dust in the alveolar walls, the septa and the peribronchial tissue still remain and there is the same connective-tissue thickening.

This histological picture of epithelial irritation and connective-tissue thickening with consequent loss of elasticity of the air vesicles easily accounts for the condition inaccurately termed "miner's asthma." This is clinically a chronic bronchitis and emphysema. We had formerly considered that the simple anthracosis could never give rise to physical signs of consolidation. Quite recently, however, we have seen an old miner who had distinct signs of consolidation down to the fourth rib on the right side. There had been no history of acute inflammation nor of tuberculosis. Repeated sputum examinations and the tuberculin test were negative and we were forced to conclude that the signs of consolidation were due to anthracosis. In some fifteen autopsies on miners we have never seen any diffuse consolidation. Only once have we made out tuberculous scars at the apices. Though these can easily be concealed in the blackened lung.

"Miner's phthisis," which is an unfortunate term used in England, is undoubtedly a misnomer as far as the phthisis goes. Real tuberculous lesions in miners are always due to the tubercle bacillus.

It may be of interest to note in passing merely as a matter of record that we have made a number of analyses of miners' lungs and those of people who are not anthracotic. By the most careful methods we could employ we found that there was a considerable margin of error. In a rough way we can say that the foreign matter in the lungs of old miners is about 33 per cent. of the dried lung. In adults who have not been especially subject to dust it runs from 1 to 3 per cent. or a little over.

The quantitative estimation of the dust in mines may also be of interest. This quantity naturally varies with many conditions. The average of twenty-five different determinations of the dust in mines which we have taken is 0.0354 gram per cubic metre. The screen-rooms in the breakers were much more dusty, giving an average of 0.3967 gram per cubic metre.

The only other quantitative determination of the amount of

coal-dust in mines which we have seen in literature is that of Hesse, quoted by Dirksen,[10] as 0.0143 gram per cubic metre of air.

A comparison of the quantity of dust in the air in mines with that in other places can be seen in the following table complied by Dirksen:

TABLE VI.—AMOUNTS OF AIR IN VARIOUS PLACES.

	Observer.	Grams per cubic metre.
City after rain	Tissandier	0 00025
Iron foundry	Arens	0 0015–0. 0280
Iron foundry	Hesse	0.0717
Saw mill	Arens	0.0150–0. 170
Snuff factory	Arens	0.0160–0. 0720
Grinding mill	Arens	0.028
Grinding mill	Hesse	0 047
Cement factory	Arens	0.13–0. 224
Felt slipper factory	Arens	0.175
Bunker on steamship	Dirksen	0.0828–2. 2897
Coal-mine	Hesse	0.0143
Coal-mine (average)	Wainright and Nichols	0.0354
Coal-breakers (average)	Wainright and Nichols	0.396

From this table it will be seen that there are a number of places as dusty as modern coal-mines. There are few places dustier than the screen-rooms of breakers.

In concluding, we wish to thank Professor Mendel, of Yale, and Professor Abbott, of the University of Pennsylvania, and Dr. Sweet, of the Rockefeller Institute, for many valuable suggestions. And the latter also for placing at our disposal facilities of the Hygiene Laboratory, where a part of our experimental work was carried on, the other portion being done in the laboratory of the Moses Taylor Hospital at Scranton.

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Cyanosis Retinæ.

CYANOSIS RETINÆ.[1]

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of Philadelphia.

This term is used to describe the changes in the fundus which are observed in congenital heart disease with general cyanosis, and was first employed by Liebreich, in 1863, in his report of a case of this kind, the illustration of which may be found in the first edition of his *Atlas*. The honor of being the first to call attention to the changes in the eye-grounds, designated by the term of "cyanosis retinæ," however, belongs to Knapp, who exhibited a water-color and described the ophthalmoscopic findings in such a case before the Heidelberg Society in 1861. Von Ammon[2] had given a short description and sketch of the exterior of the eye in congenital heart disease in 1841, but no study of the fundus had been made, as the ophthalmoscope had not yet been discovered.

Since the publication of Knapp's description other cases have been recorded, although their number is exceedingly small, in view of the comparative frequency of congenital disease of the heart. This is to be accounted for, in all probability, not by the rarity with which this form of heart disease causes changes in the retina, but rather by the failure of clinicians to examine the eye-grounds in these cases, or, in the event of such examinations being made, to allot any special significance to them.

As may be seen by the illustration, from a case recently observed by the writer, and the description of which will follow, the changes revealed by the ophthalmoscope in cyanosis retinæ are most striking, the vessels of the retina attaining a size which is rarely equalled in any other condition, this being particularly true of the veins, which may become enormously dilated and tortuous; the arteries, though usually similarly distended, being affected to a less degree. In addition to the change in their calibre, both veins and arteries become much darker than normal, the former assuming a deep-violet color, while the arteries resemble normal veins. The peripheral twigs of the retinal vessels are distended, and vessels which are usually invisible may be seen over the entire fundus. Pulsation in the vessels is absent. Small hemorrhages are of frequent occurrence in the neighborhood of the disk; more rarely larger ones are found in the macula, when vision may be much affected.

[1] Read before the American Ophthalmological Society, May, 1905.

[2] Clinical Representation of the Diseases and Malformations of the Human Eye (Tafel, vol xv. Fig. 3, Text, Bd. iii. p. 75.

As is well depicted in von Ammon's sketch, the exterior of such eyes also present marked signs of cyanosis, the lids becoming blue, the sclera violaceous, while the vessels of the conjunctiva are much distended. In the later editions of his *Atlas*, Liebreich withdrew the illustration of the case of cyanosis retinæ which had appeared in the first edition, and substituted for it one of abnormal pigmentation of the eye, to which he inappropriately applied the former name. Although, owing to the popularity of the *Atlas*, this change occasioned for a time some confusion regarding the precise application of the term, the subject is now well understood, and, as indicated in the opening sentence of this paper, the name of cyanosis retinæ is applied to the eye changes observed in cases of congenital heart disease alone.

The congenital diseases and malformations of the heart which may occasion changes in the fundi are various. Thus, they may be found where there is an abnormal connection between the right and left ventricles, and in cases in which there is a stenosis of the pulmonary artery or an abnormal origin of the aorta and pulmonary artery. They may also arise when there is a general retardation of the blood current, as a consequence of congenital dilatation of the entire vascular system.

The cause of the dilatation of the retinal vessels has been a matter of some dispute among clinicians, some maintaining that it is simply the result of the general venous stasis, while others aver that this cannot be true, as retinal changes rarely, if ever, occur in cases of general cyanosis due to causes other than congenital heart disease, and contend, moreover, that the arteries often manifest changes as well as the veins. Most authors attribute the dilatation of the vessels to a stretching in the walls of the vessels themselves, and consider this to be dependent upon a lessened resistance in the walls, due to disturbances in nutrition occasioned by insufficient oxygenation of the blood.

Although the examination of the eye-grounds in cases of congenital heart disease has not been considered by clinicians to be of particular moment, the dilatation and tortuosity of the retinal vessels being regarded as merely additional evidence of the general cyanosis, recent observations by Babinski and Nagel indicate that the ophthalmoscope may be of decided value both in the diagnosis and prophylaxis of this class of heart cases. Thus, Babinski has reported a case in which a marked dilatation of the retinal vessels preceded other evidence of general cyanosis, while Nagel, from a study of the cases in the literature, and from his own observation, asserts that if the ophthalmoscopic examination reveals that the retinal arteries as well as the veins are darker and broader than normal, the presence of a connection between the two sides of the heart is assured; when the darker coloration and tortuosity is limited to the veins, he believes that the existence of some other

congenital lesion is indicated, which does not permit the admixture of venous with arterial blood.

If these observations are true, clinicians should be made fully cognizant of them, for it is generally conceded that the diagnosis in cases of congenital heart disease is at times practically impossible from a study of the physical signs. It is hoped, therefore, that the practice of examining the eye-grounds in this class of cases may become more universal, and that the results of such examination be recorded, as it is only by means of a series of such observations that the assertions of Babinski and Nagel can be confirmed, and the actual value of the ophthalmoscope to the clinician proven.

The vision in eyes the seat of cyanosis retinæ may be normal, but if hemorrhages occur, as is not infrequently the case, vision may be more or less involved. Both eyes are usually similarly affected.

The attention of this Society was first called to the subject of cyanosis retinæ by Knapp, in 1870, who exhibited before the annual meeting of that year the sketch which he had prepared of the case exhibited by him in Heidelberg, and gave a short *rêsumê* of his notes. Since that time no further mention of the condition has been made before the Society, so that the writer considered that a brief *rêsumé* of the cases which had already been reported, and the details of two additional cases, might be not without interest.

1. As already recorded in the *Transactions* of the Society, in Knapp's case there was non-inflammatory, excessive hyperæmia of both retinæ, occurring in a patient with enlargement of the heart, but without valvular anomalies, who exhibited general cyanosis and aneurysmal bruit at different parts of the body. The retinæ were so vascular that a countless number of thick and tortuous arteries and veins arose from the optic disks and branched radiating toward the periphery, like a caput Medusæ. The area and borders of the optic disks were totally concealed. The yellow spot was distinctly discernible. A great number of arterial and venous twigs reached to the fovea centralis. The retina, refracting media, sight, and visual field of both eyes were perfectly normal. The patient died in the hospital at Heidelberg. The body was found to exhibit a general enlargement of the whole vascular system.

2. The next case was observed by Liebreich, in 1863, and, as already stated, was illustrated in the first edition of his *Atlas*. Examination of the vascular system showed stenosis of the pulmonary artery, with considerable hypertrophy of the right ventricle. There was general cyanosis. The ophthalmoscope showed the retinal veins to be twice as broad as normal, without being tortuous or presenting unevenness. The arteries were normal.

3. Leber[1] reported the third case, calling particular attention

[1] First edition of the Graefe-Saemisch Handbuch, Bd. v. p. 325.

to the color of the blood, which was unusually dark in the arteries, being about the same color that the veins normally present, while in the veins it was dark violet-brown. The vessels were full and stood out from the level of the eye-ground. The anterior ciliary veins were unusually dark and the conjunctiva cyanotic. The patient was the subject of general cyanosis, and was thought to have stenosis of the pulmonary artery. This author, in his capacity as assistant, had had an opportunity of studying Knapp's case, and referred to it at length in his treatise in the first edition of the Graefe-Saemisch *Handbuch.*

4. In 1878 Stangloneier[1] reported a case of pulmonary stenosis in which there was endocarditis of the pulmonary valves, a patulous foramen ovale and ductus Botalli, small retinal arteries and very tortuous veins, with two large hemorrhages in the nasal part of the retina, which appeared shortly before death.

5. In the next case, a man aged twenty-five years, reported by Litten[2] in 1882, the fundus was found to be abnormally dark, emitting a decidedly bluish reflex. The retinal veins also were enlarged and tortuous. Examination showed a complicated valvular defect in the ostium pulmonale, associated with a defect in the interventricular septum. The same author[3] referred to another kind of cyanosis retinæ, namely, that produced by aniline poisoning, and cited a case in which the conjunctiva and fundus appeared violet and the retinal bloodvessels almost black. Vision was unaffected.

6. Nagel reported the sixth case, occurring in a boy aged nine years. One eye was staphylomatous, following a perforating ulcer of the cornea three years previously. The fundus of the fellow-eye showed the typical changes of cyanosis retinæ, the entire eye-ground being dark red and the veins almost black. The author was of the opinion that the marked cyanosis, the dyspnœa, the coolness of the skin, the clubbing of the fingers, pointed to a stenosis of the pulmonary artery, with probable mitral insufficiency.

7. In his monograph on *Congenital Affections of the Heart* (p. 23) George Carpenter cited a case which occurred in a child five and a half years old. There was extreme general cyanosis, the face and body being dusky, the mucous membrane slate-colored and the conjunctiva suffused. Ophthalmoscopic examination "showed the retinal veins to be *very* large, the arteries of good size, and both sets of vessels most intensely cork-screwy. There was no edge to the optic disk, and the red reflex apparently started from the physiological pit." At the autopsy both ventricles were hypertrophied and dilated, the right being in excess. The right auricle was twice

[1] Inaug. Dissert., Wurzburg, 1878; Nagel's Jahrbuch f. Ophth., 1878, p. 261.
[2] Deutsche med. Wochen., 1887, No 8, p 141.
[3] Berl. med. Wochen., 1881, Nos. 1 and 2.

the thickness of the left, but the left was dilated and the right auricular appendix was four times the size of its fellow, which was decidedly small. There was a patent septum ventriculorum, capable of admitting the index finger. The foramen ovale was closed. The aorta arose equally from both ventricles, and there were further abnormalities in its branches.

8. The same author referred to a second case, seen by him, in a small boy who was somewhat cyanosed. No cardiac bruit was detected. An ophthalmological examination revealed fundal changes similar to those noted in the case just reported, and suggested to him the nature of the complaint.

At the close of his interesting monograph Carpenter gave the notes of 22 cases of congenital heart disease, but unfortunately an ophthalmological examination appears to have been made in but one instance, where the history contains the note that "the veins of the optic disk pulsate and appear beaded."

9. In Loring's *Text-book of Ophthalmology* (vol ii. p. 106) will be found the notes of an additional case, occurring in a young woman aged twenty-eight years. Loring reports: "She had never noticed that her skin was darker than that of other people until six years ago, when she was shipwrecked, during which she was much terrified. Her finger-nails were, however, club-shaped, and she had been informed by her mother that this had been the case since she was ten months old. Her skin is now of a livid, bluish tinge, with dark-purple spots upon the face and hands, and she thinks that the color is increasing in darkness. The ears are purplish and the conjunctiva a good deal injected.

The veins of the fundus are enormously enlarged and tortuous, and have an anteroposterior curve in certain places, but only on the disk and its immediate neighborhood. There are very many vessels that are plainly visible and can be traced far into the retina, which, in the normal state, are either not present or are so minute as to be invisible. The color of the blood in the veins is of a violet tinge. The arteries are considerably enlarged, but not so much as the veins. The blood in the arteries is of the color of ordinary venous blood. There is no pulsation in either arteries or veins. An exhaustive examination by an expert failed to bring to light any disease of the heart or any abnormality in its sounds. The ophthalmoscopic picture presented a strong similarity to that of choked disk, and had been, indeed, taken for that."

10 and 11. In 1892 Hirschberg observed two cases. In the first the retinal veins were dark blue, tortuous, and dilated. There were, in addition, general cyanosis, fingers clubbed, pulmonary emphysema, dilatation of right heart, and vision reduced to one-quarter in each eye. In the second case the heart was malformed and there was the same appearance of the eye as in the preceding case. Vision was normal.

12. Another case was reported by Galezowski,[1] in which vision was said to have been much lowered by intense congestion of the retina in the macular region, and, as in Stangloneier's case, retinal hemorrhages appeared a few days before death.

13. In 1899 Fehr reported a case before the Berliner Ophthalmologische Gesellschaft, April 20, 1899, but the writer has not had access to the *Transactions* of the Society, and has been unable to find the notes of the case elsewhere.

14. Quite recently Goldzieher[2] reported an interesting case of congenital disease of the heart with hyperglobulia, in which the left eye was lost, as a consequence of hemorrhagic iridocyclitis, and the right eye presented the typical changes described as cyanosis retinæ. The patient was a female aged nine years, who had always been sickly and had suffered repeatedly from nasal hemorrhages. The exterior of both eyes presented marked signs of cyanosis. The retinal arteries and veins of the right eye were much enlarged and darkened, and the edges of the disk were everywhere sharply outlined; there were no hemorrhages. The iris of the left eye, however, was stippled with small hemorrhages, and the vitreous contained so much blood that no reflex could be obtained from it. The globe was stony hard, and finally underwent spontaneous rupture through the sclera at the equator. Dr. Hochwall, who made a careful study of the case clinically, found a considerable increase in the hæmoglobin in the blood, as a consequence of increase in the number of red blood corpuscles, which he considered to be an expression of increased concentration of the blood. He thought the hyperglobulia was dependent upon the disturbance in the circulation, as a consequence of the congenital affection of the heart, and was not an instance of the symptom-complex in which there is an association of polycythæmia, cyanosis, and tumor of the spleen. The determination of the precise nature of the cardiac lesion was difficult, but the physical signs and the employment of the *x*-rays indicated that either the foramen ovale was patulous or that there was a defect in the septum atrium.

15. In 1904 Babinski[3] saw a case in a ten-year-old subject with stenosis of the pulmonary artery. General cyanosis first appeared at three years of age, during an attack of whooping-cough. Left-sided hemiplegia developed at this time also. The retinal arteries were tortuous and dilated, and the veins were enormously enlarged.

16. In a recent number of the *Annales d'Oculistique*, February, 1905, the same author, in conjunction with Toufesco, reported another case occurring in a male aged thirty-five years. The patient had been sickly, but symptoms of cardiac congestion first became manifest after a prolonged attack of typhoid fever, when

[1] No reference. Cited by Babinski, Revue neurologie, 1904, vol. xii. p. 1146.
[2] Centralblatt f. prak. Augenheilk., September, 1904.
[3] Revue neurologie, 1904, vol. xii. p. 1143.

seventeen years of age. The pulmonary artery was stenosed. *Cyanosis of the retina was found without general cyanosis, both arteries and veins being markedly dilated.* The authors conclude from this that general cyanosis as a consequence of stenosis of the pulmonary artery may manifest itself in the retina before changes appear in the skin.

17. In 1903 a case of cyanosis retinæ was observed by the writer, which had been referred to him at his service in the Howard Hospital by Dr. S. M. Hamill, who had made a careful study of the general condition of the patient, which he later embodied in a report which has been published elsewhere.[1] The abbreviated history was as follows: The patient, a boy aged nine years, first became blue when a few months old. Dyspnœa had always been marked upon exertion. At the time of examination the boy was noted as being of normal size for his age and well nourished. The skin of his face and body and all the visible mucous membranes had a deep, purplish color. The skin was rough and dry and was covered with small, bluish follicles. The hands and feet were especially congested, and clubbing of both fingers and toes was present to a marked degree. Examination of the heart showed a double impulse, which was most marked in the fourth interspace. The left chest was more prominent than the right, but there was no thrill. At the apex there was a rather soft, short, blowing, early systolic murmur, which accompanied the first part of a divided first sound. The murmur and the divided sound were both heard in the axilla, over the body of the heart, and best in the third space to the left of the sternum. The murmur was heard faintly in the aortic area. The second pulmonic sound was much accentuated. The heart's action was fairly regular and rapid. There were marked pulsations in the veins of the neck and in the subclavians. Posterior to the sternocleidomastoid muscle of the left side, and almost directly underneath the mastoid process, there was a marked pulsation, which on palpation felt like a tortuous and anomalously located external carotid artery. Dr. Hamill was unable, from the physical examination, to reach any satisfactory conclusion regarding its specific character, but inclined to the view that the case was one of stenosis of the pulmonary artery, with patulous ductus arteriosus, basing this opinion upon the age of the patient, the location of the murmur, and the greater frequency of this form of cardiac lesion.

As incorporated in Dr. Hamill's report, the condition of the eyes was as follows: The eyeballs were prominent. The conjunctival vessels, both those of the globe and those of the lids, were swollen, especially the veins, giving the eye a violaceous appearance. The pupils were large and the media clear. Vision was 5/6 in each eye.

[1] Pediatrics, May, 1903.

The ophthalmoscope showed a neuroretinitis of marked degree. The retinal arteries and veins were greatly swollen and tortuous, resembling large angle-worms. The writer does not remember ever to have seen retinal vessels of a larger calibre, the retinal veins appearing to be increased to three times their normal size. The head of the optic nerve was obscured by the swollen retina, and there were a few small hemorrhages into the nerve-fibre layer of the retina close to the disk.

18. About the same time that Dr. Hamill's case was under observation, Dr. W. C. Swindells, the office assistant of the writer, found similar changes in the eye-grounds in a case of Dr. Howard Fussell, to whom he is indebted for the following notes: "Male child, aged one year; presented no abnormality during the first two weeks of life. When next seen by Dr. Fussell, however, about a year later, marked signs of cardiac disease had manifested themselves. It was elicited from the mother that when the child was about two or three months old, and while he was lying in the cradle, he was suddenly seized by an attack of unconsciousness, during which his entire body became blue. As he grew older and moved about, attacks of dyspnœa became frequent, during which the face and fingers became blue. Occasionally, and especially when he was disturbed or attempted to run, attacks of unconsciousness supervened, during which the finger-nails became blue and the lips cyanosed. Examination revealed that the fingers were clubbed; the precordia was prominent, and there was a marked prominence over the junction of the first and second piece of the sternum. The heart dulness was at the right border of the sternum and first rib diagonally downward to the nipple line. There was a very rough systolic murmur heard all over the chest, with greatest intensity at the upper border of the heart dulness, and transmitted into the vessels of the neck.

Shortly after this, and while still under Dr. Fussell's observation, the child had an attack of what was considered to be pneumonia and later was ill with true diphtheria with laryngeal stenosis. During the treatment of the diphtheria 2000 units of antitoxin were administered, with no untoward effects. After convalescence it was noted that the heart dulness had moved still farther to the right and was broader at the apex. A month later the cyanosis was more marked and the dyspnœa had become constant. The heart's action was slow and regular. A loud, high-pitched systolic murmur was heard over the aortic cartilage. The second heart sound was accentuated. To the left of the sternum the murmur was not as loud nor the second sound as sharp. The murmur was heard, however, over all the anterior part of the chest, its point of greatest intensity being just over the sternum, below the junction of the first and second parts. It could be faintly heard in the vessels of the neck and in the back. There was no pulsation in the

external jugular. The second sound was loudest midway between the nipple and sternum, over the fourth interspace. The edge of the liver could not be plainly felt, but there was more resistance on the right side than on the left. The upper line of the liver dulness corresponded to the fifth rib in the axilla. There was no œdema of the legs. The child grew gradually worse and died a few months later with symptoms of cardiac failure. No autopsy was permitted; so that the probable diagnosis of mitral regurgitation, with, perhaps, some communication between the two sides of the heart, could not be ratified.

Owing to the fact that the diagnosis of the cardiac condition in the 18 cases which have been just cited was confirmed by autopsy in but four instances, and that the descriptions of the changes in the eye-grounds in most cases are too meagre from which to draw inferences regarding the precise lesion in the fundus which the various cardiac lesions may occasion, it has not seemed worth while to attempt to summarize the facts which have been given, or to seek to prove the truth of Nagel's assertion from them. As indicating the value of ophthalmoscopic examination in cardiac cases of this type, it is of interest, however, in addition to the case of Babinski, which has already been cited, to refer to Carpenter's second case in which there was no cardiac bruit and but slight cyanosis, and the true nature of the complaint was determined only after the discovery of the typical changes of cyanosis retinæ by the ophthalmoscope.

SCARLATINAL OTITIS.

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Strange as it may seem in these days of advanced thought in medical science, there are yet a few physicians who are firmly adherent to the old notions of tentative medicine.

After all that experience has taught as to the dangers to the functions of hearing and of life itself by ear inflammation complicating scarlet fever, yet there are many of our profession who assume an attitude of expectancy and contented helplessness, and a peculiar sense of satisfaction when an ear aches, breaks, and runs during the course of this infectious disease.

This is unfortunate for the science of medicine and for the human race, and inexcusable in these days of enlightenment.

We might excuse the doctor of past generations, who felt that scarlet fever must leave some "corruption" in the system and felt

that the organ of hearing could be spared better than the kidneys or other organs, so he welcomed with delight the appearance of pus in the auditory canal, feeling that now the system was safe from harm.

Otitis media is the most frequent complication of scarlet fever, and, everything considered, the most serious. Like the general infection, its severity varies with the epidemic, the climate, and the seasons of the year. It is more common in the winter and spring months and in the colder climates. Statistics show that from 3 to 9 per cent. of the cases of scarlet fever have ear complications, and in about 50 per cent. of these cases both ears are affected. Bezold, of Munich, collected 640 cases of aural disease secondary to scarlet fever covering a period of eleven years in which 984 organs of hearing were affected, about one-half of them being bilateral. During the same time he estimated, from other statistical sources showing 17,087 cases, that 3.75 per cent. of all cases had ear complications. In 530 of Bezold's cases 263 had a continued discharge from the ear lasting over eight years; and a total destruction of the drumhead in 25 per cent. of these cases. In another report of 185 cases he shows that in 30 there was an entire destruction of the drumhead with the loss of one or more of the ossicles; in 59 the perforation in the drumhead comprised two-thirds or more of the membrane; in 13 there were small perforations; in 44 granulations and polypi; in 15 total loss of hearing on one side, and in 6 cases total loss on both sides; in 77 cases the hearing distance for whispered voice was less than half a metre.

Of 4397 cases reported by Finlayson, otitis occurred in 10 per cent., and of 1008 by Caiger, 13 per cent.; in Burkhardt-Merian, 33 per cent. The greatest importance is attached to the inflammation of the middle ear, although the affection of the labyrinth or otitis interna are of no little moment, when one considers the large number of cases of total deafness, and the large percentage of inmates of deaf-mute institutions where otitis is due to scarlet fever.

Dr. Blau found from statistics of eight authors 14,045 cases of otitis interna with 55 cases, or 3.8 per cent., due to scarlet fever. Bruckner gives 7 per cent.

In a survey of the deaf-mute asylums of Europe, in 1887, the smallest percentage was found in Italy, 1.5 per cent., and the largest in Norway, 27 per cent., representing the ratio of the cases due to scarlet fever and showing the relative frequency of the disease in the warm and cold climates.

May has collected statistics in New York of 5613 deaf-mutes, 572 of whom, or nearly 10 per cent., owed their condition to otitis of scarlet fever. Holmes, of Cincinnati, reports that in the deaf-mute institute in Jacksonville, Ill., of 500 cases 7.2 per cent. were due to this disease. In the Rhode Island School for the Deaf out of 201 cases admitted 44, or 21.3 per cent., were due to scarlet fever.

In the Clarke School of Northampton, Mass., 105 out of 640, or 16.4 per cent., were due to scarlet fever.

On the other hand, statistics at large show that of all cases of suppurative otitis media about 12 per cent. are due to scarlet fever, thereby showing that a disease which impairs and often destroys the function of one of the most important organs of special sense, which is the focal point of infection in many cases of septicæmia, pyæmia, meningitis, brain abscess, and is a constant menace to the patient's life, is a complication of scarlet fever which is often ignored or even welcomed by the physician with content.

For illustration in the study of this disease I wish to use a season's experience in the scarlet-fever wards of the Rhode Island Hospital. Beginning about the middle of January, 1904, and continuing till the middle of June, I had the privilege of watching some 60 cases. The ears and throats of each case were systematically examined at regular intervals. During these five months 7 cases of suppurative otitis media developed, and 3 cases were in active progress when the patients were admitted, making 10 cases in all, or about 17 per cent. Seven cases were bilateral and 3 unilateral. Of the 7 which developed after admission, 4 were characterized by pain in the ear and rise of temperature and increased rapidity of the pulse. Three of the 7 had no pain whatever, the only condition which attracted attention being a sudden rise in temperature which could not be otherwise accounted for. These cases were discovered by examination of the ear, in accordance with a standing order that in the event of a sudden rise of temperature or increase of fever symptoms in any of the cases, I was to be notified immediately, it being my purpose to forestall the ulcerative spontaneous perforation of the drumhead by a good, free incision, assuring immediate depletion of the parts and adequate drainage. Of the 17 suppurating ears, 2 went on to suppuration and necrosis of the mastoid. These were operated and made an uneventful recovery. One of the cases, a girl of ten years, who had a very severe general infection, streptococcic pharyngitis and nephritis, the ear is still discharging a little at intervals.

In 4 other cases, in which paracentesis was done, the drumheads healed perfectly and hearing returned to normal condition in a very few weeks. In one of the severe cases, which had nephritis but no severe pharyngitis, the drumheads of both ears and two of the ossicles (malleus and incus) of the left ear were swept out as clean as if eaten out by acid, in spite of the most careful and energetic treatment. In this case the ears were still discharging when seen ten months later. The other 3 cases were lost track of after discharge from the hospital.

The bacteriological study of these cases was intensely interesting. The cases were very carefully followed in the laboratory by Drs. Fulton, McElroy, and Barrows.

Four cases on which paracentesis was performed showed streptococcus in pure culture in the discharge which was taken from the canal immediately after the incision was made—these were the cases which healed the most rapidly—the ear returning to its normal condition in a few weeks. All of these cases, however, showed a mixed infection within a week after the first culture. An examination showed staphylococci and other organisms with the streptococci in each case. One of these also had a diphtheria-like bacillus in combination with the others. It is interesting to note that this case healed the quickest of any case in the series, and it is also interesting to note that the case which had so extensive destruction of tissue and ossicles had exactly the same combination of organisms.

The diphtheria-like bacillus which was found in 3 cases differs from the Klebs-Loeffler bacillus, so this was not considered a diphtheria infection. I might add that while we had a few cases of the double infection, diphtheria with the Klebs-Loeffler bacillus and scarlet fever, yet in these cases under consideration the Klebs-Loeffler bacillus was not found. As yet no specific organism has been isolated as being peculiar to the otitis of scarlet fever; and bacteriology has not yet given us any specific organism for this very common infectious disease. Leutert,[1] a former assistant at Schwartze's clinic, who has done considerable research in this line, is of the opinion that the malignant forms of scarlatinal otitis characterized by rapid destruction of tissue are due to streptococcus, and he likewise thinks that streptococcus is the germ that gives to scarlet fever the picture of a severe disease, while the so-called specific germ of scarlet fever seems to have no relation to the general infection and suppuration. Kurth frequently found a kind of streptococcus laid together in chains forming a lump-like colony which he called streptococcus conglomeratus.

Before bacteriology revealed to us the possibilities of the infection in scarlatinal otitis it was always considered as diphtheritic, and nearly all authors up to the present time classify this form of otitis as diphtheritic or scarlatinal-diphtheritic; and the natural inference is that it is necessary to have a diphtheritic infection of the throat in order to have the otitis. In our cases there was no diphtheria infection in either throat or ear.

Clinically there are three forms of scarlatinal otitis which I prefer to classify as follows: 1. Acute serous. 2. Acute suppurative. 3. Acute necrotic. The severity of the otitis seems to be dependent on the severity of the general infection on the one hand, and the constitution of the patient on the other. Anatomical conditions and the structure and condition of the vasomotor system are important factors. In children previously healthy and with

[1] Archiv. f. Ohrenheilkunde, Bd xlvi and xlvii.

good firm skin, normal lymphatic system, and good resistance generally, the disease assumes the milder form if the general infection is not too severe. In children of the lymphatic diathesis, so-called scrofulous tendencies, subject to swelling of the glands of the neck, enlarged tonsils, and hyperplasia of the adenoid tissue of the pharynx, we see the severe forms of the disease.

Acute Serous Inflammation. The mild forms of otitis occur mostly about the time of the eruption or within the first ten days, or while the prodromal pharyngitis is active. The symptoms are similar to those of ordinary acute catarrhal otitis media. If the patient is old enough he complains of a feeling of fulness in the ear, pulsating noises, slight deafness, and varying degrees of pain, some slight, some severe, and in some there is no complaint of pain at all. In the young children we are only led to think of the ear by an increase of fever symptoms and evidence of discomfort by restlessness and crying; and perhaps by the discovery of a discharge from the ear.

Examination of the ear with the head mirror and speculum under a strong light reveals, in some cases, a slight redness and swelling of the fleshy portion of the canal, according to the period of the scarlet fever and the condition of the skin of the body. If desquamation has begun, the canal is usually coated with scales of epidermis, and the wax glands seem to have an increased activity at this time while the opening of the canal is usually well coated with wax. The drumhead varies from a dull, flat appearance to that of intense congestion and bulging. After three or four days of the subjective symptoms, a discharge of serum is noticed escaping from the canal, the pain subsides, the fever symptoms diminish, and if the ear receives no secondary infection it heals after a few days. Unless these cases are under close observation, as in a hospital or by a trained nurse, they are rarely seen until the drumhead has ruptured. Probably most of these simple cases become secondarily infected and continue on into the purulent form. When this is the case the discharge of serum takes on a purulent character and becomes more profuse, the patient becomes restless and more feverish, with a general increase of toxic symptoms, and suffers more or less pain deep in the ear and mastoid bone.

Acute Suppurative Inflammation. This form of otitis usually occurs somewhat later in the course of the scarlet fever than the previous form, about the second week, although it is probably dependent upon the severity of the general infection. The subjective symptoms are much more severe; the sudden increase of feverish symptoms is very pronounced, the temperature often rising to 103° or more. The pain in the ear is lancinating and throbbing in character, radiating from the depths of the ear to different parts of the head and neck. Swelling of the glands of the neck with stiffness of the muscles and torticollis are fre-

quently present in these cases. In some there is evidence of cerebral irritation shown by convulsions and vomiting, so that meningitis may be feared. We must bear in mind, however, that these cerebral symptoms are often among those which usher in the general infection, but when they occur after eruption is complete, or later, they may accompany kidney or ear complication.

Objective Symptoms. In cases where the drumhead has not ruptured, the canal is scaly and at its entrance abnormally coated with wax, and is often swollen. The drumhead is œdematous, swollen, varying in color from a bright to a dusky red, and bulging in parts or as a whole, to a greater or less degree, sometimes protruding a fourth of an inch or more into the canal. Even after spontaneous perforation has taken place this enormous protrusion of the membranes of the drum cavity is sometimes seen.

ACUTE NECROTIC INFLAMMATION. This malignant form of otitis is commonly called the diphtheritic form. This type of the disease is analogous to the throat condition known as scarlatinal diphtheria, although Klebs-Loeffler bacillus are not found in the discharges, nor are there any of the paretic sequelæ so common to diphtheria.

The term diphtheritic which infests the traditional teaching of disease of the throat and ear makes it very difficult to estimate the true conditions which have existed in the history of these membranous and exudative forms of disease of these organs; but bacteriology has done much, and we hope soon will do more, toward clarifying this befogged condition. Many cases of the so-called scarlatinal diphtheria, characterized by severe infiltration of the lymphoid tissue of the throat and membranous deposits on the tonsils and posterior and lateral walls of the pharynx and of the mucous membrane surface of the nose, show pure cultures of streptococci or staphylococci, or the mixture of the two, Klebs-Loeffler bacilli being absent.

This malignant or membranous form of otitis is to be considered the most severe form of ear disease, the infection being intensely virulent, and the destruction of tissue very rapid, often sweeping out the entire drumhead and the ossicles in a few days. If it is uninterrupted by treatment, the necrotic process destroys the inner membranes of the tympanum, those of the round and oval windows, invades the labyrinth, destroying the membranous labyrinth and organ of Corti, thus destroying the organ of hearing and disturbing the equilibration of the body causing a rolling, staggering, or tottering gait for a long time after recovery. Cases have been reported, and I have seen two, where parts of the labyrinth and cochlea were exfoliated in the necrotic process and removed as a sequestrum at operation.

In other cases the infection travels on through the internal auditory canal, damaging both the auditory and facial nerve, the latter

causing facial paralysis, and on into the meninges of the brain causing meningitis and death. Post-mortem examinations in these cases have revealed necrosis of the cochlea and deposits of membranous and necrotic tissue in the vestibule and other parts of the labryinth.

The appearance of this type of otitis, as seen through the speculum at the early stage of the inflammation, is similar to the violent suppurative form, but the drum membrane soon becomes livid, or has a yellowish-white pulpy look having the appearance of a false membrane which doubtless is present, similar to the false membrane of the pharynx, and contains quantities of streptococci. The secretion at first is not abundant, in fact very little, but in a few hours a brownish, purulent fluid with a very fetid odor of tissue necrosis is present. The hearing soon lessens, the drum membrane dissolves away, and the drum cavity is filled with this membranous exudate with a foul odor. When the labyrinth is affected its onset is signalized by sounds like the ringing of bells or the clanging of steel, and by dizziness. In some cases the labyrinth becomes involved primarily.

CAUSE. The cause of scarlatinal otitis is evidently a streptococcus infection, the method of invasion being still a matter of discussion; formerly it was thought that the avenue of invasion was through the Eustachian tube, and some hold this view to-day. More recent investigations suggest that it is hæmatogenous, the infection carriers finding their way to the tympanum through the blood current.

It seems to me that in some of the milder forms infection is carried through the Eustachian tube, and in the severer forms through the blood current or through the lymphatic system of the pharynx and tympanum, or, perhaps, like other cavities with small outlets, the Eustachian tube becoming closed by the pharyngeal inflammation, a vacuum is produced in the tympanum, causing it to fill with serum, and in the presence of infection a suppurating process is established.

PROGNOSIS. Prognosis as regards both the continuity of the organ of hearing and of life is largely dependent on prompt treatment and proper care. The simple forms are favorable, the severe ones not so favorable, especially as regards hearing, for in some a large part of the hearing is lost, and the case goes on to necrosis of tympanum, ossicles, and mastoid, in spite of the most careful treatment. Cases where adenoids in the nasopharynx and enlarged tonsils are present, also children of scrofulous diathesis, are apt to have a severe time of it. And while the outlook, as far as immediate danger to life is concerned, may be good, there is always the loss of the hearing function or its impairment to be feared, and the establishment of a chronic otorrhœa which is a nuisance to the patient and his family and friends, and with all its attending dangers it is a constant menace to life itself.

TREATMENT. *Treatment is naturally surgical and antiseptic.* We are dealing with an infectious process caused by some one or a number of varieties of micro-organisms, and this simple or mixed infection, as the case may be, must be removed as soon as possible, and the part kept as free as possible from them until healing is complete.

Some recommend preventive treatment by irrigating the nose and throat daily to keep the amount of infection reduced to a minimum. This procedure I think is one attended by great risk, as there is danger of washing infection through the Eustachian tube into the tympanum, and thereby setting up the inflammation one is trying to avoid. The patient should be kept in a warm room night and day, avoiding rapid changes in the temperature, draughts, and chilling of the body after bathing; this will do much toward preventing congestion of the ear and mucous membranes of the body, for the less the circulation in these parts of the body is disturbed the less will be the danger of inflammation. When the lymphatics of the neck show signs of swelling, ice should be applied in a throat or ear ice-bag and kept on constantly. Iodide of lead ointment is also useful in reducing the inflamed glands.

If spontaneous rupture of the drumhead takes place and the ear begins to discharge serum, this should be removed by pledgets of sterilized cotton and *not* by syringing, as any disturbance of this condition as by an unsterile syringe or other instrument might result in a secondary infection. When the discharge has become purulent then it should be syringed every two or three hours, in order to keep the canal as free from pus as possible, and avoid an infection of the deeper parts and the external glands of the ear and lymphatics surrounding it.

When a case of scarlet fever is first seen by the physician, instructions should be given to the parents or nurse regarding the ear complications, and if the patient complains of pain in the ear, or manifests any discomfort in the ear by putting the hand to it or rubbing it, moving the auricle, or refusing to lie on the affected side, or if a sudden rise of temperature occurs, strict orders should be given that the attending physician be notified at once; and he should immediately examine the ears, and if any of the appearances of the drumhead above described should be present, he should at once incise it freely. If the drumhead is to be incised, the canal must first be sterilized by syringing with 1: 2000 bichloride and carefully dried with sterile cotton, and a sterile instrument used. The canal should then be closed by sterilized cotton, and changed as often as it becomes saturated, great care being taken to see that the hands and instruments are clean. If the discharge becomes purulent, the canal should be syringed and any one of the following solutions may be used: Sodium bicarbonate solution 1 drachm to the pint, lime-water, normal salt solution, carbolic acid,

1: 40; saturated solution of boric acid, or 1: 500 permanganate of potassium solution. After cleansing, the canal should be dried with sterile cotton and a solution of boric acid in 60 per cent. alcohol instilled into the canal. In the severe cases a 1 per cent. solution of bichloride of mercury in 60 per cent. of alcohol, or 2 per cent. nitrate of silver solution may be dropped into the ear, these solutions having been previously warmed.

If swelling of the mastoid lymphatics or tenderness over the mastoid region occur, the aural ice-bag should be applied, and if relief is not had within a few hours, leeches should be applied over the tip and upper part of the mastoid bone. These measures will usually give prompt relief and often stay the progress of the inflammation. If the disease goes on to suppuration of the mastoid, operation will be necessary, and should be performed without delay; yet if the indications for operation are not especially urgent, I prefer to wait until after desquamation is complete, as in my experience if the operation is performed during desquamation, or if within six or seven weeks from the onset of the fever, the repair process is extremely slow, even after a week has elapsed following operation, there will be little or no formation of new tissue in the bone cavity. The discharge from the wound is very profuse and intensely acrid, excoriating the parts wherever it touches, ulcerating the fresh cut surfaces and sloughing out the stitches and destroying the new healthy granulation tissue that may have formed. The large open wound resulting from this destructive process affords an unprotected area for septic infection, and with the virulent nature of the discharge, which contains quantities of streptococci and staphylococci, the system is exposed to great danger. One of our cases developed a pronounced septicæmia, unquestionably from this cause.

A word regarding the contagious nature of the discharge would not be amiss. A recent experience at the hospital will furnish a good illustration of the care needed in handling a discharging ear immediately after the scarlet fever has had its run. There were three children representing three families who had fulfilled the demand of the Board of Health regarding quarantine and returned to their respective homes, each child with suppurating ears. Within ten days after their discharge from the hospital one other child from each family was admitted with scarlet fever, and it is believed, after careful investigation, that these new cases were started by coming in contact with the discharge from the ears of the children who had just returned home. I believe that the discharge from a scarlatinal otitis serves as a good infection carrier, and cases should be detained in an intermediate station, and from other children at least two weeks after desquamation is complete; during this period the ears should have thorough antiseptic cleansing. As a safeguard to the community I think it is essential to consider the

discharging ears in the determination of raising quarantine in scarlet fever cases.

In conclusion, let me emphasize the importance of early recognition of the ear complications of scarlet fever; we should not expect and wait for the ear to "break and run," as is so often done, for this is but little short of criminal negligence, but, through prompt treatment by paracentesis of the tympanum as soon as there is the slightest indication of inflammation in the tympanic membrane or exudate in the tympanic cavity, relieve the distressing symptoms and place the patient in the safest possible condition as regards systemic infection and intracranial complications, and do all in our power to preserve the function of one of the most important organs of special sense.

CONCERNING THE CAUSES OF GALLSTONES.[1]

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My purpose in presenting this short paper to this Society is twofold. First, I wish to indicate a new method of investigating a very difficult problem, the causation of gallstones, and to show what results this new method of investigation has borne in my hands. Secondly, I want to interest the members of this Society, who are in a position to examine organs derived from a large number of autopsies, in this method of investigating, so that more cases may be brought together and studied, and the correctness of my data be either verified or denied.

Without going into the earlier history of the theories of gallstone production, which would lead us far astray and tend to obscure the points that I wish to emphasize, I would like to unfold to you the development of the present theory of gallstone production. This development is closely bound to the Strassburg school of medicine, to Naunyn and his pupils. The work of this school gave us our present conception of the etiology of gallstones.

Before the publication of these modern views the whole question of gallstone production was unsettled—just as unsettled as the questions of the etiology of pulmonary consumption prior to Koch's discovery. Diathesis, heredity, age, sex, were regularly named as the causes of cholelithiasis, while the actual processes at work were scarcely considered. In the early nineties Naunyn attacked these

[1] Read before the New York Pathological Society, March, 1905. Since reading this paper I have been able to add a new case to my series.

time-worn causes and he applied all his energies to the annihilation of the diathetic theory and to the building up of a new theory based on bacteriological facts. As was perfectly proper and logical, he set to work to determine the origin of the substances found in gallstones. Bile-pigments, cholesterin, and the organic framework had to be studied and investigated. He had to determine whence they came, under what conditions they were produced, and whether they were increased or diminished by changing the conditions of the experiments. In this work, Kausch, Thomas, and Jankau were of the greatest assistance. From the data furnished by these experimenters and investigators, Naunyn concluded that (1) the solids in bile are independent of general body conditions, that cholesterin is a product of the bile passages, and as such is not influenced by any diathetic or non-local conditions; that (2) bacteria enter the bile system, set up an inflammation, catarrhal in type, which blocks the free outflow of bile, and which is accompanied by an increased production of cholesterin, from the inflamed mucous membrane of the gall-bladder, and *pari passu* an exudation of albuminous substances. In this bacterial catarrh all the elements of the gallstones are present and by processes of precipitation and infiltration stones develop absolutely independently of the general body conditions.

When this view was published no convincing experiments were put forth to establish the positive fact that infection of the bile passages actually produced gallstones. That work was reserved for others and a number of years elapsed before this evidence was furnished. Mignot and Mijake succeeded in producing gallstones experimentally in animals by first partially occluding the cystic duct, thus producing incomplete stagnation of the bile, and then introducing bacteria into the gall-bladder and causing an inflammation of this organ. This work gave the firm support that Naunyn's theory had lacked when it was originally published. Since Mignot and Mijake did their work, others have repeated the experiments with varying results, but no one seems to have tried to follow and analyze the genesis of stones in the human body.

It seemed to me that if certain cases could be studied, I would be in a position to trace the development of gallstones in the human body. To obtain the same or similar conditions to those of the experiments of Mignot and Mijake, I would have to get livers in which there was stagnation of bile and infection of the biliary ducts, as the gall-bladder in acute inflammatory conditions rarely comes to the post-mortem room unopened. Patients are either operated and the bladder is emptied or they get over the acute attack and we know not what has occurred in the bladder. The few that die of this condition represent the most acute cases, and, as they are of short duration only, the study of stone formation is practically impossible because of the early stage of development of the same. These facts turned my attention to the liver and its ducts. Here

we find obstruction and inflammatory conditions combined; here in cases of common duct obstruction and secondary cholangitis the conditions obtained by the animal experiments are paralleled. In these cases, which unfortunately for this investigation are rather rare, nature reproduces the conditions that Naunyn thought underlay the process of gallstone production. The conditions are purely local; there is stagnation of bile and there is inflammation of the mucous membrane of the bile passages. If it were possible to obtain a sufficiently large number of human livers with the above conditions present, the correctness of Naunyn's views, as well as the direct applicability of the results of animal experimentation, might be verified and the theory of gallstone production in human beings would rest on a firm basis.

I was able to collect 12 cases at autopsy[1] in which there had been a common-duct obstruction for more than four or five weeks, and in which a more or less severe inflammation of the extra and intrahepatic ducts had developed. In 7 of my cases the common duct was obstructed by calculi, in 5 by tumors of various kinds. *In the first series of 7 cases, as a result of the conditions of the experiments stones formed in the liver-ducts. In the second series of 5 cases, though apparently the same conditions were present, no stones developed, though stagnation of bile and inflammation of the mucous membrane of the bile passages were present.* In this second series Naunyn's causes were present just as in the first series, but no stones developed. How are we to explain this difference? Why should gallstones be produced in the liver-ducts in one series and not in the other? Is it mere chance that cases with previous cholelithiasis regularly showed stone formation in the liver-ducts, when common-duct obstruction due to stone and cholangitis developed, while those that had no previous stone formation did not develop stones, when stagnation of bile and cholangitis developed?

The absolute regularity in the results seems to me to preclude chance, and even though my series of cases be small, I think it points to a new factor in the production of gallstones. It is needless to theorize at this stage as to what this factor may be. If, to make my point clearer, we call the third factor an altered liver metabolism or a diathesis, we could understand why stones developed in the liver-ducts when stagnation and infection developed in cholelithiasis cases (they having the predisposition as evidenced by the older stones), and why they would not develop under similar conditions (second series) where no liver disturbance or diathesis obtained. But the term diathesis is vague and almost meaningless, merely a garb for ignorance. Perhaps, however, a faulty hepatic metabolism underlies the formation of gallstones?

[1] Similar cases of the clinical surgical literature have been brought together by me in a paper entitled "Intrahepatic Cholelithiasis," Medical News, 1904.

A larger material bearing on this subject could readily be gathered by the members of this Society without entailing any great trouble, and a larger material would help to clear up this difference that I have described between the cases of old cholelithiasis and those without cholelithiasis. And, as I said at the outset, it was with the purpose of enlisting your assistance that I read this paper.

In closing I may sum up the results that I have obtained as follows:

1. *Naunyn's factors—stagnation of bile plus inflammation of the bile-passage mucosa—do not seem to be sufficient by themselves to lead to gallstone formation, even though the time allowed for the working of the causes be adequate.*

2. *My first series of cases shows that these two factors lead to stone formation in patients who previously had gallstones. In this series we have the first real evidence of the factors underlying gallstone production and the causes of cholelithiasis in human beings.*

A CONTRIBUTION TO THE DOCTRINE OF THE MORPHOLOGY OF THE BLOOD, DERIVED FROM EXPERIMENTAL TOLUYLENDIAMINE POISONING.[1]

By Privat-docent Ernst Schwalbe, M.D.,
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and
John B. Solley, M.D.,
of New York.

The advances made in staining methods in recent years have increased the range and certainty of our classification of changes in the morphological elements of the blood. This was notably the case after the application of Ehrlich's method, a fact which may have led that author to state so optimistically that the doctrine of the changes in the blood, in as far as it depends upon morphological investigations, especially with the aid of color analysis, belongs to the, for the greater part, enlightened chapter of general pathology, and appears to have reached, provisionally, a definite conclusion, so that, essentially, only a few points remain to be cleared up.

Against this encouraging view is the constantly increasing list of contemporary works dealing with the blood morphology from clinical, pathological, and anatomical standpoints. As example, one

[1] Based upon a series of experiments conducted under Dr. Julius Arnold at the Pathological Laboratory of Heidelberg in 1901.

Schluss-betrachtung zu der Leukaemia, Bd. viii. der spec. *Pathologie u. Therapie*, Wien, 1901.

needs only to mention the diversity of opinion still held as to the genesis of erythrocytes and leukocytes. Furthermore, the terms "immunity, hæmolysin," etc., of rather recent coinage, suggest possibilities of undiscovered facts just over the borderland of morphology and intimately related to the latter. It seems difficult, therefore, to exclude the possibility of morphological conditions and changes, too fine to be detected by our present technique, occurring in the blood under the influence of toxic substances, bacterial poison, etc.[1] In conclusion, it is to be noted that even the firmly established doctrine of leukæmia and "typical anæmia," as based upon clinical evidence, is constantly meeting with opposition, in the light of more recent investigations.

In undertaking the following experimental study with toluylendiamine, we were impelled by the belief that blood morphology is still an unexhausted field containing details important for our knowledge of clinical phenomena. The mode of attack was influenced strongly by the strikingly analogous phenomena in the physiological process of coagulation as compared to the changes occurring in the blood in various anæmic conditions. It was Alexander Schmidt who pointed out this analogy.

In studying the process of coagulation, he produced artificial anæmiæ experimentally and compiled facts of intense interest and importance. To follow with understanding the points to be discussed, presupposes an accurate knowledge of the morphological processes in coagulation.[2] The necessity of being familiar with the physiological changes in the blood in judging of pathological processes is self-evident.

By employing toluylendiamine, the authors hoped to produce an artificial anæmia that might bring light to bear upon some questions of urgent clinical importance; also to have an opportunity to study such an anæmia throughout its course. In many cases of anæmia there are morphological changes to be indicated in two directions: of degeneration, of regeneration. As is well known, the differentiation is often very difficult, in fact, thus far well-nigh impossible—*e. g.*, the nature of the basophile granules in the red cells. The analogy presented by the phenomena of coagulation is encouraging, however.

Some authors maintained that many poisons produce typical morphological changes.[3] Comparing the result of our experiments with those of other investigators and inducing here the earlier results

[1] For example, the ring-like structures recently found by Cabot (Journal of Medical Research, February, 1903) in the red cells with Wright's modification of Leischmann's stain.

[2] Schwalbe. Ueber Blutgerinnung, 1900.

[3] This view was held by Heinz, Limbeck (Wiener klin. Wochenschrift, 1893), and Parlier, and opposed by Maragliano and Castellino (Zeitschrift f. klin. Medizin., Bd. xxi.). Ehrlich also expressed himself against Heinz (Ueber schwere anaemische Zustände, Verhandlungen der XI. Congress f. innere Medizin., 1892).

attained in studying coagulation, a further explanation of the conclusions to which we were lead seems possible.[1]

Through the agency of toluylendiamine, we hoped to bring new material to the question of the province of the blood plates; to get closer to the structure of the red cells, and to ascertain, to a degree, at least, the significance of the hæmoglobinæmic "inner bodies;" and finally, to verify or refute conclusions derived from earlier investigations.

Toluylendiamine[2] was first employed by Stadelmann to study icterus. His first conviction that the blood was not affected by the poison was later relinquished after Afanassiew's demonstration of the marked changes in the red cells. The drug was then used by many as a poison and Afanassiew's observations were confirmed. The action of the toluylendiamine varies greatly in different animals. As a rule, canines form the best subjects for study; moderate dosage is followed by marked icterus and high-grade changes in the blood. Hæmoglobinuria is the exception, in distinct contrast to the effect in cats, in which hæmoglobinuria is intense and almost constant, the icterus being secondary and often absent. In dogs, if the dosage is given carefully as to amount and time interval, the icterus soon disappears and the animal becomes active. In rabbits prolonged dosage can be effected without the appearance of icterus or hæmoglobinuria, but often with the production of a typical picture of severe anæmia. Rabbits, furthermore, tolerate enormous doses, especially by carefully graded increase. Guinea-pigs react much the same as rabbits; at least we have never observed icterus in either. In dogs a picture similar to that observed in rabbits can be obtained by working up gradually from very small incipient doses—namely, icterus is absent, but a severe anæmia develops. Icterus would, therefore, seem to be produced only by large single doses.

The effects of acute, subacute, and chronic poisoning were studied by means of fresh and dried blood specimens, treated by numerous staining methods. As material for investigation, chiefly dogs were used (six dogs, four rabbits, and two guinea-pigs). As to methods, there seems to be no question that wrong or one-sided conclusions can be avoided only by employing many methods and carefully comparing results. For fresh specimens, Arnold's Holland plättchen-

[1] Since the compilation of this article the views of many investigators have changed, and bear out our previous views against the specific action of various blood poisons. Bloch (Deutsch Archiv f. klin. Medizin., 1903, Bd. vii. p. 277, cited by S. Batty Shaw, *Practitioner*, vol. lxxii No. 4), for example, in studying pernicious anæmia concludes that the same "must be due to some agent which interferes with the activity of the blood-forming organs," and found no evidence to support any toxic nature of this agent, "whether arising from disturbed internal secretory activity of any organ, or from digestive disturbance due to atrophy of the mucosa of the bowels, or to the occurrence of bacterial invasion." He also found that ingestion of toluylendiamine produced nothing more than secondary anæmia.

[2] Discovered by Hoffmann in 1861 as a secondary product in making aniline dyes.

method proved very satisfactory. Bettmann's method of using neutral red gave interesting results. To fresh specimens, many coloring agents were applied, neutral red and methyl violet being most satisfactory. In using the latter, however, the Holland disks have to be discarded on account of the attraction of the walls of the meshes for the coloring matter. To obviate this, one can employ Neumann's method, which consists in spreading the mixture of blood and coloring agent between a cover-glass and smaller square portion of a cover-glass and inverting the former upon a smeared hollow-ground slide. The dry specimens were fixed in the usual manner; heat, alcohol, ether, formaldehyde, formaldehyde vapor, etc., are all equally reliable when properly employed. At first numerous staining methods were tried, but were finally abandoned for Laurent's solution of eosin methylene blue, Engel's method (*Zentralblatt*, 1900), and hæmatoxylin and eosin. With Ehrlich's triacid stain the results were not satisfactory.

A detailed report of the records seems superfluous and liable to prove monotonous, being prolonged in some cases over months. Also a tabulated comparison of blood examinations, as first planned, would add nothing in clearness. The essential is a classification of the morphological changes observed and a description of the individual groups with illustrations. The literature of toluylendiamine poisoning can be dispensed with here for brevity's sake, careful study and review of the same being understood; also, all histological and chemical changes, such as were discussed in considering the processes of coagulation. That any such study as the present requires careful macroscopic and microscopic examination of all the organs as well, is self-evident. In the following we are concerned, however, only with a description of the blood as studied morphologically in fresh and dried specimens:

I. Changes in the red cells.
 Blood counts in anæmic conditions. Degeneration. Regeneration.
 a. Phenomena of budding. Process of expulsion of parts of red cells. Question of the blood plates.[1] Intracellular hæmoglobinæmic bodies.
 b. Other phenomena of degeneration.
 Poikilocytosis. Polychromatophilia. Microcytosis. Macrocytosis. Shadows.
 c. Granules in the red cells.
 d. Appearance of nucleated red cells in the blood and loss of nucleus in same.

[1] The classification of blood plates under red cells, and not separately, is not in accord with many recent articles on the subject, but is the result of conclusions stated elsewhere (Schwalbe, Anatomische Anzeiger, 1900, Bd. xx.), for it is not possible to regard the blood plates as other than products of the blood cells, especially of the red cells.

II. Changes in leukocytes.

Results of counts. Granules in white cells. Changes in the white cells not being recognized as referable especially to the action of toluylendiamine will be treated secondarily.

The various investigators are agreed, as a glance at the literature will show, that more importance is to be attached to the changes in the red cells than to those in the leukocytes. That the red cells are markedly influenced by toluylendiamine is clear from the clinical course of the chronic poisoning, in which all the features of a high-grade anæmia are present, intense emaciation and striking pallor of all visible mucous membranes. The blood count as obtained by Biondi and Bernhardt, also by Heinz, confirms the clinical picture. The blood counts made during the present study correspond in every case, the unusual oligocythæmia being readily recognized. One source of error should be considered, however; as mentioned in the literature, the red cells of poisoned subjects have a greater tendency to disintegrate, that is, a diminished resistance. It is certain that the conditions under which cells are counted further diminish their resistance, as a result of which the count obtained may be lower than is the case. Even with this factor considered, the oligocythæmia is sufficiently positive to be notable. By means of the count the possibility is excluded of the strikingly pale blood being caused somehow solely by decrease in the quantity of hæmoglobin.

One of the most interesting phenomena observed in the red cells is connected with the formation of the blood plates, the budding of the erythrocytes. The process is studied most satisfactorily in fresh blood specimens from a dog freshly jaundiced by toluylendiamine when examined in the Holland disks as described above. The common "thorn-apple" forms are seen in great numbers. Presently the budding process begins, the buds varying in size. A protrusion forms upon some part of the red cell, the part affected often showing a lustre differing from the rest of the cell. The projecting part becomes hemispherical, and, often with amœboid movements, gradually becomes more detached from the cell till the uniting pedicle divides the "pre-existent" blood plate.[1] Two points worthy of note are that from the outset of the study the number of blood plates and amount of budding were greatly in advance of the same observed in normal blood, and that an extensive destruction of red cells took place in fresh blood while under observation. The inference is that the same sort of disintegration takes place as in coagulation, only much more rapidly under pathological conditions. In

[1] It is to be clearly understood that the movement of the bud in this process is not to be confused with the Brownian movement of particles, or attributed to capillary attraction or currents in the serum. The recent studies of surface tension by J. Traube (Pflüger's Archiv, December 1, 1904) bid fair to ultimately explain many, if not all, of the phenomena of amœboid movements and disintegration (budding of the blood plates, etc.) of the morphological elements of the blood.

other words, the resistance of the red cells is greatly lessened. An observation should be noted, which, without leading to definite conclusions, presents the possibility of significant deductions for the morphology of coagulation, namely, that in a dog in a condition of chronic poisoning at a time when, without icterus, a marked anæmia was present, surprisingly few changes were found in the blood in fresh specimens. This suggests invitingly the conception that in such cases the less resistant red cells succumb, the more resistant alone being left in the circulation. Its ultimate connection with the process of coagulation can merely be indicated. It is known that in artificial anæmias—*e. g.*, by repeated venesection, the coagulability of the blood at first increases, then gradually diminishes; also, that this decrease can reach a serious point. No attempt was made, in fact, no method is extant, to determine the rate of coagulation, so that any conclusions are impossible. These facts are of apparent interest, however, in assuming the possibility of some connection between these morphological changes and the process of coagulation.

In toluylendiamine poisoning the budding process is, therefore, apparently greatly increased, although this cannot be stated as a fact from simple observation of fresh specimens, especially when it is known that the budding process occurs with some irregularity in coagulation and that in toluylendiamine poisoning no method is known by which to accurately determine these changes. By admixture of some coloring fluid—*e. g.*, methyl violet salt solution, as described elsewhere,[1] as emphasized recently by Bloch and Heinz, the changes become more significant. Applied to normal blood, no coloring is visible at first in the red cells. As the budding process begins, many of the buds become more or less deeply colored with methyl violet. The red cells send out plump projections, staining blue in the same manner that the "pre-existent" blood plates stain with this fluid. After prolonged observation many red cells appear to contain a granule stained blue more or less deeply, as described elsewhere.[2] These blue granules seen in the red cells of normal blood resemble entirely the blue granules described by Heinz. In the blood of animals poisoned with toluylendiamine, the process is quite different. The blood from a dog jaundiced with toluylendiamine presents a beautiful picture; immediately after withdrawal the red cells are almost uniformly occupied by blue granules and blue projections, the greater number having only one visible blue granule; some having several. On careful inspection it is evident that all of the blue particles are projections; that they are the same as those seen in studying coagulation. Fig. 1, *a* to *e*, shows several of the forms seen immediately after withdrawal; many red cells have

[1] Schwalbe. Münch. med. Wochenschrift, 1901.
[2] Schwalbe. Anatom. Anzeiger, Bd. xx.

retained their form; some contain unstained as well as stained projections. All, or nearly all, of the red cells present this picture in the blood of jaundiced dogs; in rabbits or dogs which, though repeatedly poisoned, remain unjaundiced, the number is fewer. These phenomena are interesting in several respects: First, they challenge to a comparison with other poisons; Heinz, who has determined the effect of many poisons, found the same manifestations produced by some of the poisons when studied with gentian violet salt solution. He called the stained particles in the red cells "blue granules." Bloch regarded these blue granules as products characteristic of pyridin poisoning, and affirms that he did not find them in cases poisoned with tolylendiamine. That the "blue granules" of Heinz and blue bodies (Körperschen) of Bloch are identical with those found by us can be derived from the drawings of these two authors. This would seem to indicate that these phenomena are not characteristic for any particular poisons,[1] a point made more clear by comparison with the processes observed in coagulation, in which, in normal blood, the blue granules appear in the same manner, but in fewer numbers, and only after coagulation has commenced, whereas, in poisoned blood the granules are seen immediately on withdrawal. In both instances, therefore, we have apparently to deal with an injury of the erythrocytes, a degeneration, the difference in the blue granules and projections being quantitative rather than qualitative, the degenerative process in cases of poisoning occurring after the type witnessed in coagulation. It is possible, of course, that all poisons do not act upon the red cells in the same way, but produce types of degeneration too subtle for the present technique; the rapidity and energy of the injury to the blood cells is apparently of the same importance for the production of the morphological picture of poisoning, however, as the nature of the poison.[2]

Some few poisons act by choice upon the erythrocytes; others cause the production of methæmoglobin. The distinction is evident. In staining with methyl violet salt solution possibly the blue granules predominate in some forms of poisonings, whereas, in others (still beyond our power of detection) they might be much less numerous and conspicuous. If, however, there were changes in the red cells in a sense characteristic of certain poisons, or which only appear under the action of a particular blood poison or chemically allied substances, no evidence of such was found in these investigations, or is presented in the literature (Ehrlich, Limbeck, Maragliano, Castellino, and others). It seems further impossible, in view of the fact, as stated, that all degenerative processes in the

[1] In Heinz's more recent publications, the conception of specific changes for certain poisons is emphasized less strongly than in his earlier works.

[2] Ehrlich said in regard to the changes described by Heinz: "Personally I am of the opinion that in higher animals this degeneration will be produced by the majority, perhaps by all, of the blood cell poisons." (Verhandlungen d. XI. Congr. f. innere Med., p. 411.)

red cells, so far as it is possible to determine them with or without the aid of staining fluids, find their prototype in the process of coagulation. It remains to explain the relationship of the phenomena witnessed in studying coagulation and those obtained more recently, by means of methyl violet salt solution, to the "inner bodies" of Ehrlich (Hæmoglobinaemische Innenkörpenschen).

In fresh specimens the red cells are often filled with granules which have a stronger lustre than the rest of the cell. On closer inspection these are seen to be projections which are markedly hæmoglobiniferous. An unequal distribution of hæmoglobin in the structure of the erythrocytes in the most diverse ways is met with in fresh as well as dried specimens. Ehrlich regarded the hæmoglobiniferous "inner bodies" as the result of a degeneration of hæmoglobiniferous parts of the cell. In dried specimens the projections, which in the fresh specimens stain deeply with methyl violet, are eosinophilic, as direct comparison teaches. In fresh and dried specimens made simultaneously from an animal poisoned with toluylendiamine, the former show beautiful blue projections uniformly throughout the field, the latter (stained with eosin-methylin blue or hæmatoxylin) only occasional, faintly blue-tinged projection, among the red cells. In the one case they are, therefore, strongly basophilic; in the other eosinophilic, and occasionally also faintly basophilic. These qualities, taken into consideration with the altered appearance of the projections in unstained fresh specimens, point strongly to degeneration of the hæmoglobiniferous part of the cells. This is Ehrlich's view also, for in his publications he affirms the identity of the granules described by Heinz in his study of poisonings, with the "inner bodies." Conditions closely related to, if not identical with the "inner bodies," are present without question in toluylendiamine poisoning, and the transition to blood-plate production is a slight one, as will be explained. In dry specimens the projections are often beautifully shown, and of various sizes. Naturally conservatism is necessary in regard to the value of the appearances in dry specimens, as similar changes are seen as artefacts in the process of preparation. When, however, such buds are seldom seen in the preparation of hundreds of dry specimens from normal blood, in contrast to their almost constant presence, uniformly scattered throughout the field, in the blood from toluylendiamine subjects; when, furthermore, one can demonstrate the agreement of fresh and dry specimens, it would seem permissible to utilize dry specimens in the analysis of the findings.

Blood plates occur in dry specimens in clusters in the well-known manner; also separately, often in surprising numbers. Also the forms known as blood disks, with and without the presence of hæmoglobin. Stained with methylene blue or hæmatoxylin and eosin, the blood plates sometimes show a slight metachromosis, a tone partaking of the blue of the basic stain and of the pink of eosin. A

"nuclear" structure is often apparent, stained exquisitely blue, as described earlier by Arnold, and as emphasized recently by Deetjen especially (who, to be sure, gives another significance to it). Some of these plates, mostly of a delicate form, appear as if caught in amœboid movement in the preparation of the specimen, discrete or attached to a red cell, and with the nuclear structure stained blue (Fig. 2). That they are identical with Deetjen's findings is evident from a comparison of the drawings.[1]

Since Hayem and Bizzozero called attention to the blood plates in the seventies (they had been noted casually before by many authors) these bodies have claimed greater interest. The two essential questions in regard to them, their origin—thereby the question of pre-existence—and their significance in the processes of degeneration and coagulation, are still unsettled. Three views are extant as to their genesis; that they are thrombocytes (Dekhuysen), that is, independent pre-existent bodies in the blood, having the physiological importance of separate structures and constituting an essential third element of the blood, requiring explanation as to development and origin; that they are not pre-existent, but merely artefacts, globulin sediment and products of precipitation—an opinion with few retainers at present; that they are derivatives of the blood cells, either of the red cells, or white cells, or both. For a long time the leukocytes were regarded as the sole source by many eminent investigators. An extensive list of publications show, however, that the red cells are regarded as sharing at least in their production.[2] The participation of the blood plates in the process of coagulation has been observed repeatedly since Hayem and Bizzozero, and its significance variously interpreted. The works of Arnold, Franz Müller, Feldbausch, and Schwalbe should be mentioned in this connection. From prolonged observation of the budding of the erythrocytes in fresh specimens, with and without the admixture of methyl violet salt solution, it is difficult not to connect this phenomena with the increased production of blood plates. This was also the observation made by Arnold, and later by Schwalbe, in studying coagulation, and which led to the same conclusion, namely, that the blood plates are buds from the erythrocytes. As previously stated, blood plates were found in large numbers in fresh and dry specimens from poisoned dogs. In dry specimens the blood plates often found attached to the erythrocytes can be explained in two ways: either that a bud has been caught in the act of emerging from the red cell—a process often seen in fresh specimens—or that a blood plate has wandered to the red cell and become adherent—a process that unquestionably also occurs frequently. But to determine in the dry

[1] Virchow's Archiv, Bd. clxiv.

[2] Interesting in this connection is Stengel's excellent review of the recent literature (Progressive Medicine, June, 1902), and his views in support of the latter theory, published after the initial compilation of this paper.

specimen between the two is hardly possible. One feature is striking, however: in Fig. 2 red cells are seen with a dell at the point of attachment of the blood plate, giving the impression of a loss of substance at that point. In such cases it may not be too daring to assume that the loss of substance is due to exodus of the blood plate at that point. Furthermore, comparing the fresh and dry specimens: in the dry specimens the supposedly budding blood plate contains a nuclear-like structure stained distinctly with hæmatoxylin, the "nucleoid" of Arnold, the nucleus of Deetjen, Dekhuysen and Kopsch.

Fig. 2, *h*, shows a blood plate with nucleoid, and in addition, hæmoglobin stained with eosin. This observation, namely, of blood plates that contain a nucleoid body staining with hæmatoxylin or methylene blue, and that are also hæmoglobiniferous and stainable with eosin, has been seen repeatedly, not only in the pathological specimens, but also in normal blood. Unless this be purely adventitious, it speaks clearly for the erythrocytic origin of the blood plates. It should be emphasized again that the findings are taken into account only in support of this view and that the comparison of fresh and dry specimens is very striking. The conviction that the blood plates are divided from the blood cells is founded not only upon the phenomena studied in fresh and dry specimens under normal and pathological conditions and upon the observations made in following the process of coagulation; it is also verified by Arnold's investigations of circulating blood, especially his extensive study of thrombi treated by the most diverse methods. The objections to the deductions of Deetjen and Kopsch and the opinions of Hirschwald have already been published.[1]

The usual changes present in anæmic conditions were also found in toluylendiamine poisoning, namely, the so-called shadows (to which Afanassiew referred especially in connection with toluylendiamine poisoning), poikilocytosis, and polychromatophilia. In how far the latter is to be regarded as an indication of degeneration is beyond discussion here. The striking variation in the size of the red cells, especially the presence of very minute forms, is noteworthy.

As to the granules in the red cells: basophile granulation of the red cells was described at an earlier period by Askanazy and others, but not until after the studies of A. Plehn did any great number of investigators begin to turn any particular attention to them. Among the more recent work should be mentioned that of Grawitz, Litten, Hamel, Bloch, Isserlin, Cabot, Fisch, and Stengel. Plehn, whose views are compiled and beautifully illustrated in the monograph of 1901, believes that certain basophile granules of the erythrocytes, found in the blood of malarial patients subsequent to recovery, are

[1] Schwalbe. Anat. Anzeiger, Bd. xx.

to be regarded as latent forms of the plasmodium. It is interesting that later, after Plehn's view was rather generally refuted, Bloch, after examining Plehn's specimens—having previously disputed Plehn's contention—came to the conclusion that the granules in Plehn's specimens were with great probability of a different nature from those described by himself and others, and that therefore Plehn's view could not be rejected without further proof. If Plehn's view is correct, it presupposes the existence of two distinct kinds of basophile granules; one characteristic of latent malarial parasites alone; the other common to, if not constant with, the most diverse forms of anæmia; the anæmia of Biermer, of carcinoma, of different toxic conditions. It is difficult to reconcile the present literature with Plehn's view, though only a systematic study of malarial blood would justify any conclusions.[1]

The granules seen in toluylendiamine poisoning correspond throughout with those described by Grawitz, Litten, and others. In dog's blood they are not constantly present. Guinea-pig's blood gives the most beautiful granules, both under normal and pathological conditions. In normal blood of healthy rabbits the granules are also present, but less frequently than in guinea-pigs. Bloch states that he did not find them in normal blood of guinea-pigs. Engel found them in the red cells of embryonal mice. In the guinea-pigs studied there was a marked variation in the size of the granules, both in the polychromatophilic and otherwise normal red cells. In dog's blood, in which, as stated, they were present only in poisoned cases, the variation in size was even more marked. They were most frequent in the chronic cases, rare in acute cases. The large granules are to be regarded apparently as "nucleoid" (Fig. 3, *c-c*). These resemble the nucleus of the blood plates described by Deetjen. In Fig. 3, *a*, *b*, *f*, and *k*, the granules or nucleoid is surrounded by an apparent concentration of hæmoglobin, that is, a "hæmoglobinæmic

[1] Since this paper was first drafted an opportunity was offered in June and July, 1902, to study a series of over thirty cases of malaria in New York. They were all tertian, six were double. In only two cases were granulations present in addition to a frequent poikilocytosis and polychromatophilia. In all others no granulations were found even weeks after recovery. Jenner's and Laurent's stains were used, the latter bringing out the variations in color in the manner produced by Wright's modification of Leishmann's stain. The two cases presenting granules in the red cells were in adults with a double tertian infection of three and four weeks duration respectively. In the specimens from both cases numerous granulated red cells were found containing in addition young hyaline bodies (Figs. 25, 26). The granules corresponded throughout with those seen in toluylendiamine poisoning, in some instances being large and fewer to a cell, in others smaller and more evenly distributed, especially throughout the periphery, with a tendency to ring formation. One of these cases presented normoblasts, and among them cells like those reproduced in Fig. 3, *i*, and Fig. 4. In neither case could hyaline bodies be found resembling in any way the large granules in Fig. 4; and, vice versa, one could not easily mistake such large granules for even the youngest hyaline bodies. These large granules correspond in size, form, and staining qualities, however, to the granules described and reproduced by Plehn as latent malarial organisms, and were found repeatedly, as mentioned, in the blood of toluylendiamine subjects. In the two cases cited, the large granules disappeared after administration of quinine, just as in the dog after cessation of the toluylendiamine dosage.

inner body," with nuclear substance. Prolonged observation of such forms raises the question as to whether they may be preparatory forms of blood plates in the stage previous to expulsion; at any rate, the indication is not easily disregarded.

As to the smaller granules, two views exist as to their province; that they are products of nuclear degeneration of nucleated red cells; that they are the expression of degeneration of the hæmoglobiniferous protoplasm. It is not inconceivable that they are evidence of regenerative processes. Bloch, who discusses these phenomena at some length, regards them as evidence of protoplasmic degeneration. One fact against the view that they are connected with nuclear degeneration is that they are found in red cells whose nuclei are undergoing mitosis (Fig. 12). Until further evidence can be produced, however, Bloch's view must remain unchallenged.

In mitosis the entire cell is concerned, protoplasm and nucleus both being involved in the process of division. It seems inconceivable that nuclear degeneration is to be inferred from the presence of granules in a cell whose nucleus is in a condition of growth. The same argument is to be brought with scarcely less probability against protoplasmic degeneration. Assuming that the protoplasm and nucleus of a dividing cell are under the same conditions, the protoplasm must also be in a state of productive activity. The view is not untenable, however, that the nucleus, in preparation for mitosis, rids itself of certain useless material which is manifested in the shape of granules. The same may be true of the protoplasm. Bloch's findings present nothing definite for or against nuclear or protoplasmic degeneration or even the possibility of some other significance. The deductions of Engel, establishing these granules as nuclear fragments, are at least not disproved by Bloch.[1]

In studying the process of coagulation in normal blood, basophile granules were found, which stained with methyl violet and varied in size like those seen in fresh specimens in toluylendiamine poisoning, the latter being mostly large and regarded as being associated in some way with the production of blood plates. The finer granules found in dry specimens cannot be classified unqualifiedly with the above. Whether they are similar to the finer granules seen in fresh specimens when stained with other fluids—*e. g.*, neutral red, is an open question beyond discussion here.

As to the granules being nuclear fragments or products of degeneration of the hæmoglobiniferous protoplasm or capable of other

[1] Cabot (Journal of Medical Research, February, 1903) thinks the granules found in various anæmic states may be due to over-ripeness of the cell or to degeneration of the protoplasm. But with Schmidt and others he believes that they are derived from the nucleus, and not from the protoplasm, whenever changes occur in the nucleus of a degenerative or regenerative character. The ring-like structures found by Cabot in some cases of pernicious anæmia, lead-poisoning, and lymphatic leukæmia mentioned in a previous foot-note, are regarded by that author as having some possible connection with the granules, and as probable derivatives of the nucleus in the same way as the basophile granules. Unlike the latter, however, they do not stain with hæmatoxylin. No special significance is attached to them.

interpretation, a uniform explanation, in the sense that only one or the other possibility is to be admitted, seems improbable. Both may apply. Still another possibility is that cited by many investigators, especially Arnold, namely, that the protoplasm of the red cells may have a granular structure, hidden by the hæmoglobin under ordinary circumstances. Clinical experience speaks fully for the existence of changes in the hæmoglobin leading to destruction of the same without disintegration of the cell form, as, for example, in the so-called shadows. One is often reminded of this possibility by appearances observed in the red cells in fresh and dry specimens, and of the further possibility that such conditions may be revealed to us by the aid of finer technique.

In toluylendiamine poisoning, as in every case of anæmia the course of which is not ended rapidly by recovery or exitus, the process of degeneration is accompanied by an attempt at regeneration. It has been established that even after intense icterus the animal can recover. Nucleated red cells are found as an expression of this attempt at regeneration. Ehrlich classified normoblasts, megaloblasts, and microblasts. In toluylendiamine poisoning normoblasts were preponderant, megaloblasts being rare. It is well known that a differentiation depends more upon nuclear characteristics than size, and that it can at times be very difficult to distinguish between the several forms. In rare instances very minute nucleated forms have been found (Figs. 20, 21) that must be regarded as microblasts. Further description of the various forms observed is not essential here (Figs. 5 to 21). Many can be classified as normoblasts by the nuclei apart from the size. Some nuclei contain a more deeply stained nucleolus. In some instances mitosis is present (Fig. 16); in others two discrete nuclei are found in a cell and the protoplasm "laced in" between them (Fig. 18). This observation of mitosis in the circulation has been made repeatedly. Triform division of the nucleus has also been seen in the specimens.

Index to Illustrations.

Fig. 1,[1] *a–e*. Dog V. Red blood cells as observed in fresh specimens. Methyl violet salt solution.

Fig. 2, *a–p*. Dog I. Blood plates with nucleoid, in part free, in part attached to red cells: *c, f, g*, show cell at point of attachment; *h*, red blood cell with blood plate containing nucleoid and hæmoglobin stained with eosin. Hæmatoxylin and eosin.

Fig. 3, *a–k*. Different-sized granules in the red cells, hæmatoxylin and eosin: *c*, the large blue-stained granules represent the "nucleoid" of the blood plate, also in *d* and *e*; *i*, nuclear degeneration and fine granules; *j* and *k*, fine granules and a large granule.

Figs. 4–8, 10–12. Dog VI. Nucleated red cells with granules. Laurent stain.

Fig. 8. Polychromatophilic nucleated red cell with granule.

Fig. 9. Guinea-pig. Nucleated red cell with granules. Laurent.

Fig. 13, *a, b*. Dog II. Nucleated red cells. Hæmatoxylin and eosin.

Fig. 14, 15, 16. Dog I. Nucleated red cells. Hæmatoxylin and eosin.

Fig. 22. Expulsion of nucleus.

Fig. 23, *a, b, c*. Dog IV. Small lymphocytes. Eosin methylene blue.

Fig. 24. Normal red cell for gross comparison.

Figs. 25, 26. Red cells containing granules and hyaline malarial organisms.

[1] Fig. 1 drawn by Schwalbe, the rest by Solley.

FIG. 1

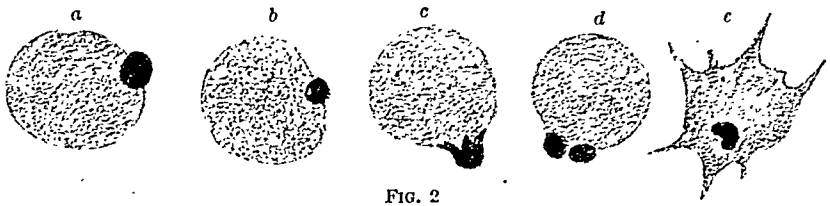


FIG. 2

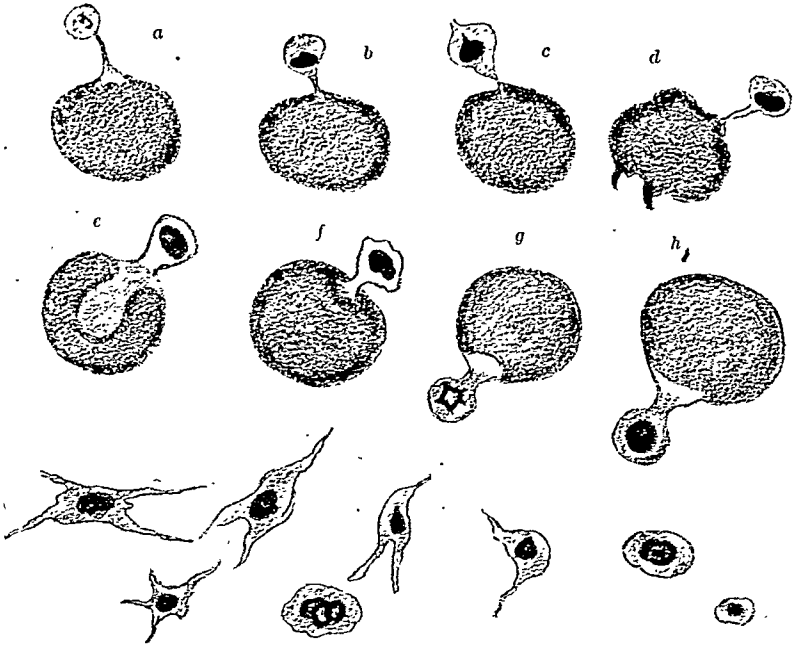


FIG. 3

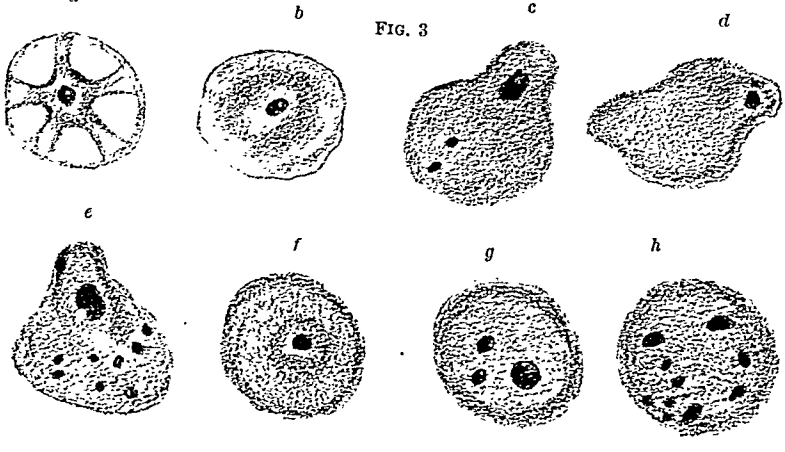
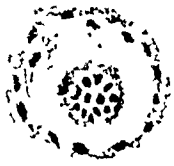
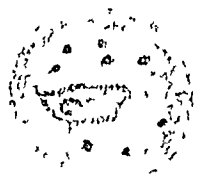


FIG 3

FIG 4







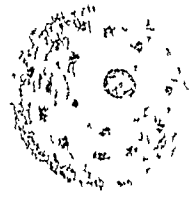


FIG 5

FIG. 6

FIG. 7

FIG. 8

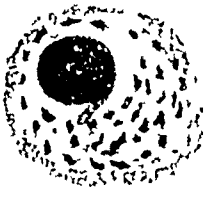






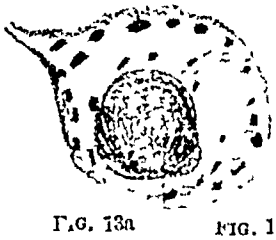


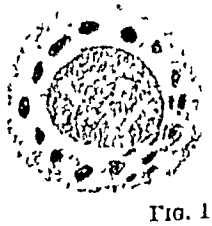
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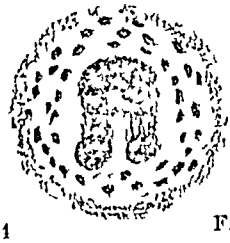
FIG. 10

FIG. 11

FIG 12







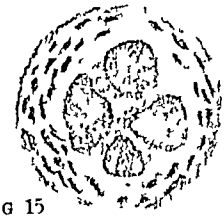


FIG. 13a

FIG. 13b

FIG. 14

FIG 15



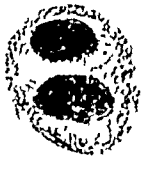






FIG. 16

FIG. 17

FIG. 18







FIG. 19

FIG. 20

FIG. 22

FIG 24







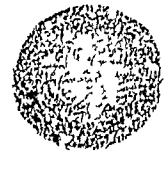


FIG. 21

a

FIG. 23

b

c

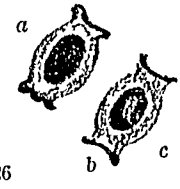




FIG. 25

FIG 26





The question as to how the nucleus of the red cell disappears can be little influenced by the present study. There were numerous instances of free nuclei in the blood and of others in the act of expulsion (Fig. 22). Disintegration seems to go hand in hand with expulsion, but it is impossible to determine how much of this is natural and how much artificial. The relation of the granules to nuclear degeneration has already been discussed. An instance of nuclear degeneration is shown in Fig. 3*i*, one of many found in the blood from a dog during acute poisoning. In fresh specimens the nuclei stain rapidly and beautifully with methyl violet. There is no apparent advantage in Bettmann's method of employing neutral red, applied by that author in studying the blood of embryonal mice.

That the white cells are little affected by toluylendiamine is shown by observation, as well as a study of the literature. Accurate counts usually denoted a slight leukocytosis, never a hypoleukocytosis. The increase must generally be regarded as relative, however. Biondi claims to have found a leukocytosis to twice or thrice the normal count and regarded it as absolute. In the present observations such counts were rare and cannot be regarded as absolute, in view of the impossibility of employing a counting method that will exclude errors due to disintegration of the red cells and the constantly changing proportion of morphological elements to serum under the influence of the toluylendiamine dosage. There were no positive indications at any time of degeneration of the leukocytes, although the nuclei were often strikingly pale. A peculiar form of small lymphocyte (Fig. 23, *a*, *b*, *c*), found in several cases, is worthy of mention. Interesting granules were found in the leukocytes, which could not be referred to specific action of toluylendiamine, and which could not be considered here without regard for the enormous literature, that would exceed the present purpose. Hirschfeld, in his comparative investigations, concludes that the polynuclear eosinophile leukocytes are common to all mammalia, but that every species possesses in addition polynuclear leukocytes with specific granules. The human species possesses in its neutrophile granular leukocytes a specific form found in no other animal. In poisoned rabbits, beside eosinophile and pseudoeosinophile leukocytes, certain forms were found with large red granules, which, by reason of their arrangement and unusual size, could not be classed with eosinophiles. Others were found with violet granules of varying sizes, sometimes varying in the same cell, often crowded together throughout the protoplasm. The doctrine of the granules is such a disputed one as to make impossible any attempt to discuss the findings of the present study. The staining quality of the granules alone does not apparently present a fundamental distinction, but rather stands in direct relation to the condition of the cell function. (Arnold; Hesse, Virchow's *Archiv.*, Bd. clxvii.)

In conclusion, the changes found in the pathological processes produced by toluylendiamine poisoning are the same as those observed in many other toxic and anæmic conditions and find their analogy essentially in the changes seen in the study of physiological degeneration. If the purpose of these pages is accomplished, they will serve as a help or incentive to others to use these observations and deductions in studying and attempting to shed new light upon the manifold and complex questions of blood morphology.

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XEROSTOMA.

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WHILE a relative degree of aptyalism, or more correctly partial suppression of the buccal and salivary secretion, is not uncommon as a result of the administration of atropine, or occurs during the course of various infections such as typhoid fever, and is also dependent upon long-continued mouth breathing from obstructing adenoid masses in the nasopharynx, yet as an independent affection *per se* this condition of xerostoma, or dry mouth, is exceedingly unusual.

Symptomatically, the affection consists essentially of a great diminution or almost complete arrest of both the secretion of the salivary glands and the numerous mucous glands of the buccal interior, while the symptom-complex thus produced evinces such a clear and definite picture that but little basis exists for confusing it with any other condition. What may be designated relative xerostoma is also found, in addition to the conditions previously mentioned, in diabetes and as a physiological state in old age to a moderate degree, while a somewhat analogous condition has been described by Heerat as psychic salivation. In four cases of the latter, which he studied, there was continual spitting without apparent cause, the patients considering it necessary to get rid of the mouth secretions. The saliva then became repugnant to such a degree that it was no longer swallowed, and as a result of this psychic state the glands were at first stimulated to increased secretion, which the patient constantly expectorated; while as a later phenomena dryness of the mouth resulted. In true xerostoma, however, while the excessive dryness is the most prominent symptom, yet other evidences of a more serious nature are associated with it and, as in the few cases recorded since it was first recognized by Hutchinson, the affection seems to deviate but little in every instance, it occupies

a somewhat limited field by itself; while, as no constant histological lesions were found in the cases that have come to autopsy, the etiological factors concerned in the production of this affection still remain obscure.

The various theories that have been evolved in this respect are most diverse, yet all seemingly having an underlying basis of a nerve origin of the disease. In some the first appearance of the xerostoma seemed to bear a close relation to a fright, an hysterical condition, malnutrition, worry, or mental overwork, while in a case seen by Shurly locomotor ataxia was present. It has been suggested that the affection is the result of derangement of some problematical centre governing the glands of this portion of the alimentary and respiratory mucosa, as in not a few of the reported cases coincident trophic changes were also found in other portions of the body; so that these cases at least strongly imply either a central change or a neurotic affection involving in part the peripheral trophic nerves. In the case reported by Harris there was complete arrest of the salivary and buccal secretion, and, in the absence of any apparent organic disease, he regarded the xerostoma as a functional neurosis; while, from the records of 12 other cases, he believes that it is not dependent upon any disease of the parotid glands, but that it is due to an affection of the nerve supply of all the glands of the mouth.

In this connection the case reported by Chappell is of great value. The affection was seen in a female forty-six years of age, and in in addition to the mouth dryness, the eyes, nose, larynx, and trachea were also involved in the same manner. Four years after the onset the parotid glands would intermittently swell, become tender, and again in a few days would subside to their normal condition. The dryness of the mucosa was complete, continuous, and the lining of the buccal cavity was sprinkled with small particles of food which adhered to the surface and produced a constant desire to drink and clear the mouth. As the teeth decayed within a year after the appearance of the first symptoms, they all had to be removed; later both parotid glands were much enlarged, nodular, hard, and non-sensitive; while the submaxillary and the lowest of the posterior chain of the cervical glands were also enlarged. Medication, locally or internally, had no appreciable effect in producing mouth secretion, and the taste was markedly impaired. As the result of the death of a friend, she suddenly lost the use of the left arm and leg, but after being confined to bed for some time there was a partial return of power. The parotid enlargement then increased, and a few weeks later she became drowsy, could only be aroused to take nourishment; then complete paralysis of both arms and legs occurred, followed shortly by death.

The nervous element in this case, of course, was well marked, more so than is usually found; but among reported cases three had a rapid onset, one a severe mental shock, another was in an hysteri-

cal woman with anuria, while still another was a hypochondriac. It has also been suggested, without any definite basis, however, that the condition of the mucosa results from the loss of certain unknown constituents of the saliva, but this theory appears somewhat problematical, although, with further studies made in the chemistry of the mouth and pharynx secretions in relation to various local conditions, this problem may in time assume a clearer aspect than it does at present.

Xerostoma is essentially an affection of middle age or later years, and is exceptional in those under forty. It is also found mostly in the female sex and, with but few exceptions, males have not seemed to be affected with the condition. As exceptions to this age at which it first appears, are the cases seen by Riesman and Osler. In the former's patient the woman was thirty-eight years old, and, in addition to a dry, pasty feeling in the mouth and throat, she had burning pains along the course of the inferior maxillary nerve. The tongue was red, bare, and studded with petechial hemorrhages on the under surface. While in the case seen by Osler, that of a male aged thirty-two years, which seems to be the youngest reported case, there was a peculiar growth along the alveoli, consisting of remnants of food which had collected, become hardened, and adherent as the result of the absence of salivary and buccal secretion.

The case which came under my observation was that of a male, German, aged, seventy-six years. No occupation. He complained of excessive dryness of the mouth, pharynx, and larynx, with constant spitting a material of a pasty character, and resembling boiled starch. His family history was negative, both parents living to old age; his sister being well at eighty-one years, while his two children died at an early age of tuberculosis. At twenty he had gonorrhœa, but gave no history or evidence in any way of syphilis. At irregular intervals, from 1870 to 1872 inclusively, he was in a hospital with prurigo (?), and the itching was intense for a period of about three years. Had chronic cystitis and enlarged prostate for twelve years, but this no longer concerns him at present. Other than this he has been perfectly healthy until four months previous to when he was first seen, when he had a slight sore throat, and this was followed in a few hours by the sudden development of excessive dryness of the mouth.

Examination at his first visit showed a tall, large-built man, apparently in perfect health, and of unusually strong physique. He walks several miles daily for exercise without fatigue, but gets extremely nervous and trembles without cause. As he has tried a multitude of remedies for his mouth condition, without obtaining the least benefit, his nervousness has increased and at times he becomes melancholy, so much so that on several occasions he has considered the question of suicide. He is a rapid talker, although his voice is normal, and he shows a marked nervous element when

he speaks. He is now devoting his entire time to the condition of his mouth, and greatly fears that the affection is incurable and progressive and that it will ultimately kill him. He rests normally at night, has no trouble to get asleep, but when he is awakened his lips and mouth burn. His memory has failed to a considerable extent, but no gross nervous changes are demonstrable. His temperature and pulse were both normal, and remained unchanged during the entire time that he was under observation. He has always used alcohol in moderate amounts, but now it makes his mouth dry and burning, although he still takes a small amount before breakfast, but does not touch it during the day. His teeth commenced to decay several years before the xerostoma developed, and about a year previous it became necessary to extract all the remaining stumps and wear a plate, which he uses constantly, as the mouth feels better when it is in place. Before the present condition developed he weighed 165 pounds, but now his weight is 146 pounds and he states that the constant worry over his condition has broken down his health, but his general appearance belies this. He complains of a sweet taste constantly present, the lips pain and sting, while the tongue feels dry and sore to some extent. His mouth is not as dry at night as during the day, as during the latter time he has to make an attempt at expectoration every ten or fifteen minutes, at night he only has to do it once or twice. On some days, but rarely and with no appreciable cause, the dryness will not be as annoying as at other times, and on account of this dryness he has a constant desire to clear his pharynx and mouth of the very small amount of thick, starchy material which takes the place of the saliva.

Examination shows olfaction to be normal; the middle turbinals on both sides of the nasal chambers are slightly enlarged and all the tissues of the nasal interior show well-marked evidences of sclerosis. The nasopharynx and pharynx are sclerotic and devoid of moisture, while the larynx interior presents the same appearance. The mouth is exceedingly dry; the mucosa has a shining appearance and a very small amount of moisture is present, so that the tissues are not absolutely dry. At the sides of the tongue, where small pools of saliva are normally present, they are absent, or consist at the most of a few white, tenuous bubbles. The palate and buccal walls respond feebly to the touch of the sharp probe, but motion is normal except from the restriction imposed by the absence of the saliva. The tongue is fissured, dry, and more or less covered with a layer of desquamated epithelial cells which proliferate and make up the bulk of what may be considered the saliva. The glands of the mouth and pharynx secrete a small amount of thick, viscid mucus, which from the admixture of epithelial cells is white in color and from the constant attempts at expectoration is seen in the mouth as firm elastic bubbles. The reaction of the saliva is acid. The right parotid gland is slightly enlarged and lobulated, the left is

normal in appearane, while both submaxillary glands are slightly enlarged. As regards taste, sweet and saline show no change from the normal, but acid is only recognized after contact for several minutes and bitter is also appreciated after a considerable lapse of time. The urine has a specific gravity of 1012, acid reaction, no sugar, no albumin or casts, but contains considerable bladder mucus and other than this is normal.

As far as treatment was concerned, nothing used was of any permanent value and after a lapse of six years the condition remains unchanged. At various times he was given at bedtime pilocarpine, gr. $\frac{1}{24}$, dissolved on the tongue, which produced a slight increase in the secretion, but this rapidly became useless and no better results were obtained with $\frac{1}{12}$ grain. Potassium iodide, commencing with 5-grain doses and increased to 60 grains, produced no beneficial effects even after several months of use. Menthol in strong solution in a hydrocarbon oil applied locally several times daily gave also negative results, while at this time my notes show that no change has occurred in his condition and nothing that has been done for him has had any effect upon the xerostoma. Fluid extract hydrastis, 5 drops in water, was used as a mouth wash, and 3 drops were given internally three times daily with no change in the mouth and an increased dryness of the laryngeal mucosa. At this time he became very much depressed over his condition and within three months had lost twenty-five pounds in weight, while nothing that had been done for him had been of the least benefit. He was given undiluted peroxide of hydrogen to use as a mouth wash as required, but its first application produced such an excessive effervescence from the enormous amount of desquamated epithelial cells present in the mouth that it was diluted 1 to 3 parts with water, with the result that he never had anything to help him to such an extent. After a few weeks' use of the hydrogen peroxide the mouth still remained dry, but the desquamated epithelium had in great part been removed and the mucous membrane looked much cleaner than before. It was not long, however, before this failed and he was given menthol crystals to place on the tongue as often as the dryness became intolerable. Under this and other local and general medication, the condition from time to time grew worse or remained unchanged, although his general health did not seem to be greatly impaired; the upper respiratory tract appearing practically the same as when he was first seen, but the tissues showed more of an atrophied aspect, especially the larynx.

This case presents to a very close degree the usual group of symptoms seen in this affection, and it seems somewhat remarkable that the symptom-complex should be so similar in practically every case so far recorded, very little variations in this respect occurring in all instances. The case recorded by Sharp shows this similarity

very clearly, differing in but few essentials from the one forming the basis of this paper. The affection occurred in a woman of forty-one years and had lasted for eighteen months before she came under his observation. The buccal dryness came on suddenly after a nervous shock and the condition was worse when she was out-of-doors or near artificial heat. The odor of the breath was musty, chewing and swallowing had to be aided by sipping water frequently, while the mucosa of the lips, mouth and tongue was dry and glazed, dryness, however, extending only to the base of the tongue and not involving the mucosa of other portions of this tract. The tip of the tongue was red, the remainder was pale in color, while the papillæ were prominent. The salivary glands did not secrete but to a slight extent and the small amount of moisture occasionally present was slightly alkaline in reaction.

In not a few of the cases the general health remains unimpaired for a long period of time, the degree of the xerostoma apparently bearing no relation either to constitutional impairment or even to changes in the digestion, as is well shown in my case where the dryness was excessive, but the digestion was at no time deficient. In other instances the lessened secretion may be prominent as regards the nasal mucosa and also the lacrymal apparatus, and instead of beginning in the buccal cavity it may first be noticed in the former regions and later involve the mouth, pharynx, larynx, and trachea. The color of the mucosa is usually lighter than normal, or may present a bluish, congested appearance; it is uniformly dry and smooth and the absence of gland tissue macroscopically is very striking. Aside from the dryness of the buccal and adjacent mucous membranes, the tongue presents the most marked features, as it is usually red, lacks its epithelial coating in part, is fissured to a greater or lesser extent, and may have the appearance of a piece of rough leather or even of alligator skin. Anteriorly the papillæ are absent in cases of long duration, but the circumvallate papillæ remain unchanged. The mucosa of the buccal cavity and oropharynx may at various points be quite sensitive to the touch of a probe, while in other portions it is more or less anæsthetic, but these conditions vary from time to time and do not bear any fixed relation to the distribution of the nerves of this region. This hyperæsthesia of the mucosa when seen in a case of long duration, may interfere to some extent with deglutition, but as a rule, if swallowing and articulation are interfered with, it is the result of the dryness of the parts; while the taste is markedly diminished, or entirely in abeyance from the suppression of moisture in this region. Should the xerostoma be extensive there may be some impairment of the voice, an irritating cough may cause considerable annoyance, and gastric disturbance may occasionally arise, although it seems remarkable that the digestion remains but little impaired even when the salivary secretion is practically *nil* for long periods. In nearly

all the cases reported the urine was negative and organic changes of the kidneys or other organs were absent.

Among other prominent symptoms excessive dryness of the skin has been occasionally noted, while quite constantly the affection has been preceded or accompanied by dental decay and the gradual crumbling away of the teeth, until finally the patients would loose all their teeth. This latter phenomena is well shown in a case communicated to Chappell by Richardson, of a female, sixty years of age, who, after a severe nervous shock, developed headache, insomnia, and attacks of intermittent deafness. Three years before she first came under observation her teeth began to crumble away, so that nearly all had to be extracted. She then developed excessive dryness of the mouth, and on account of this found difficulty in swallowing food, so that she had to drink water frequently to aid in deglutition. Hall in a study of 39 cases of this affection obtained details of the disease sufficient to be of value in 36, and of these, 32 were females. The mucous membrane of the nose was involved in 10 cases, that of the eyes in 7, and the skin was dry in but a small number. There was crumbling of the teeth in a large number; while in several the onset was sudden, in 6 it followed severe shocks, and in most of the cases the cause was unknown. While nervousness and some degree of physical impairment is not unusual in cases of long duration, insomnia is occasionally complained of and the patient practically always exhibits great fear that the affection is malignant and incurable, this being a prominent symptom in my case.

As regards the implication of the salivary glands, the author last referred to found in 36 cases that the parotids were enlarged equally or unequally in 8; in 3 they were painful and tender; 1 gland ulcerated into the mouth; in 5 the increase in size varied from time to time, and in 1 of these it was most marked at the menstrual periods. He also found that with but one exception the other salivary glands were not enlarged. In cases where the parotid glands have been enlarged the ducts have remained pervious and unchanged, and one may be fairly accurate in stating that there are no constant pathological changes thus far ascertained of these glands. As a further study of some of the symptoms, Frazer found in 19 cases that 13 had dryness of the mouth alone, 3 had dryness of the mouth and nose, 1 of the mouth and eyes, and in 2 the mouth, nose, and eyes were all involved. In 5 the teeth were absent from the first symptom of the xerostoma, but in 1 the teeth were well preserved.

After the mouth dryness in a severe form has once been inaugurated, it practically remains as long as the patient lives, but it seems to reach its maximum intensity within a short time and then remains with but little or no change for an indefinite period. The duration of the affection is therefore long and indefinite and appears to have

but little tendency toward shortening life, as in my case it has existed for over six years. In a case seen by Lartigau, in a man of sixty-four years, the affection appeared a few weeks after his wife had suffered a tragic death. The salivary glands were normal in size, taste was diminished, perspiration had decreased after the salivary secretion had disappeared, and treatment was absolutely ineffective; while three and a half years after the case was first seen the condition had undergone no change and the mucous membrane of the mouth and pharynx was extremely dry, so that considerable discomfort was caused in swallowing.

While an occasional case has been reported in which local or constitutional remedies have for short periods apparently been of some benefit in temporarily relieving the xerostoma, yet such relief was of short duration and ultimately failure along therapeutic lines seems to be the inevitable result. This is shown in a case seen by Robinson and reported by Chappell, of a woman with xerostoma following *la grippe*. There was pain in the roof of the mouth to one side of the median line, so that at night she was obliged to place a piece of paper against the painful spot in order to prevent contact with the tongue, which rendered the pain much worse. She had to drink water frequently at night to relieve the buccal dryness, but the urine was normal. Chlorate of potash and strophanthus made the dryness worse, while oil of wintergreen in olive oil applied locally gave temporary relief. At a still later period, after a number of remedies had been used, she was slowly growing worse in spite of all treatment.

Galvanism and pilocarpine have seemed to give the best temporary results in the majority of cases, while for the constant dryness of the mucosa of the upper respiratory tract aqueous or oily solutions containing various medicinal substances may be employed. With pilocarpine the best results—and even these are but transient—have been obtained by allowing the patient to dissolve $\frac{1}{8}$ to $\frac{1}{20}$ grain on the tongue, especially at bed-time; or he may employ it more frequently in the same manner in doses of $\frac{1}{100}$ to $\frac{1}{200}$ grain. Potassium iodide, glycerin, chamomile, and a host of internal and local remedies have been employed with little or no benefit; while Shurly obtained some effects from chloride of sodium held in the mouth and bismuth boroglyceride applied to the mucosa by a brush. The same author states that acacia, tragacanth, and balsam of Peru are occasionally useful; while internally strychnine, fluid extract piscidia erythrina, and tincture chamomile may also be employed.

CLINICAL INVESTIGATIONS OF THE DIGESTION IN THE INSANE:

A CONTRIBUTION TO THE STUDY OF THE NEUROSES OF THE STOMACH, WITH SPECIAL REFERENCE TO HYPERACIDITY AND BITS OF MUCOSA FOUND IN THE STOMACH WASHINGS.

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The following pages record the results of a series of investigations upon the work of the digestive glands in the insane conducted at the Michigan Asylum for the Insane at Kalamazoo, Dr. William M. Edwards, Medical Superintendent. The work was begun in the spring of 1900. A short and very incomplete report of the earliest part was published in the *Proceedings of the Michigan State Medical Society* of 1900, and the *Physician and Surgeon* of the same year. Because of other pressing duties, it was impossible for us to devote our time exclusively to this study, accordingly a number of the reports are not as complete as we would wish and the work has been drawn out over a considerable length of time. This extension of time, however, has been of distinct advantage in a few instances, as it has enabled us to gain a better idea of the more or less constant condition of the secretions of the gastric glands.

The suggestion for our work was received from von Noorden's article, "Stitungsbericht der medicin Gesellschaft zu Giessen," published in the *Archiv fur Psychiatrie und Nervenkrankheiten*, Band xvii., 1887. As a somewhat careful search through the literature failed to disclose any verification of von Noorden's work, we came to the conclusion that should we attempt to duplicate his researches so far as possible for purpose of comparison, we would not be forcing an entirely unneccssary investigation upon the medical journal and its readers. It would be difficult to improve upon the manner in which von Noorden conducted his researches; our report differs from his in only a few details, and these we will indicate as we go along.

It goes without saying that the neuroses of the stomach are encountered more frequently than any other group of stomach diseases, at least in America. Indeed, it is to be questioned whether we do not encounter an associated neurosis of some kind in a large percentage of the true organic diseases of the stomach, such as chronic gastritis, peptic ulcer, and dilatation. It is impossible to multiply too many facts concerning the neuroses, and we trust that

others will take up the work where we have left off, improve upon it, and investigate the condition of the stomach functions in other mental states. Work of this character can be carried on much better by someone who resides at an asylum, where his patients are constantly under his control and observation. The asylums owe a debt to the profession in this particular. Cases should be studied more carefully than we have been permitted to do. The relation of appetite to the secretions, the effect of changing mental states upon the secretions and functions, the postmortem findings, and especially the study of the gastric mucosa—which has been preserved by filling the stomach with formalin solution immediately after death—should be investigated. The mucosa in particular offers a promising field for research. In no other field of medicine is there such a dearth of pathological knowledge as in insanity. Lastly and very important is the psychological study of these peculiar phenomena of digestion. This we do not attempt, but leave it for those who, by special training, are better fitted for this task.

The writers are greatly indebted to Dr. William M. Edwards, Medical Superintendent of the asylum, for placing at their disposal the patients and laboratories of the institution. To Drs. Inch and Barrett, as well as to Dr. Cowie's former assistant, Dr. Ralston Williams, acknowledgment is also due for valuable services.

HISTORICAL. *A Brief Résumé of the Investigations that have Been Made on the Secretions in the Insane.* So far as the subject of this contribution is concerned, the history is a brief one. We have only to go back eighteen years for the older ideas concerning the secretions of the stomach in melancholia. Until this time alienists were content to consider the various gastric symptoms complained of by melancholiacs, and they are many, to be due to a gastric catarrh or nervous dyspepsia, and that the loss of appetite and its consequent symptoms were due to a diminished secretion of the gastric juice. Naturally from such reasoning, hydrochloric acid and pepsin were extensively used in the treatment of these cases.

For some reason von Noorden's work seems to have been overlooked by alienists in general. A perusal of the more popular works on insanity would at least lead one to this conclusion, as no mention is made of this valuable acquisition to medical science. When, on the other hand, we turn our attention to the domain of internal medicine, we find that, particularly in those works devoted more or less exclusively to diseases of the digestive apparatus, von Noorden's researches in this field are well known and frequently quoted.

The study of the urine in the insane has been considerable. Bondurant may be said to be foremost among those who claim that albuminuria is of frequent occurrence in the insane, independent of morbid histological changes in the kidneys. In a large number of these cases the albumin was detected by tests that are far more delicate than those employed in the usual urine examinations, and

it has been claimed that control experiments upon normal urines have not been sufficiently conducted. It is thought by some that the albuminuria in the insane is similar to that which can be induced experimentally. Albumosuria is thought by Pilcz to be of more or less constant occurrence in mania, and peptonuria, glycosuria, and acetonuria are thought by others to have some bearing on the insane state. Just what all these various phenomena mean is certainly thus far by no means comprehensively explained, and little, if any, practical value has been derived therefrom.

The study of the effect of the so-called autointoxications which are brought about by the absorption of ptomaines and leukomaines resultant upon the improper splitting up of albuminous substances in the intestinal canal has received the attention of a number of investigators. Experiments have been conducted, the object of which has been to test the effect of the injection into animals of urine and stomach contents from the insane and from cases of autointoxication. The toxicity of the material injected is thus estimated. Toxic substances in the blood of the insane have been demonstrated by Naegeli and his followers by a reaction which occurs when certain algæ (spiro-gyra) are placed in diluted blood; but little if any importance has been attached to this, as in all disease states more or less toxic substance is being constantly carried in the blood and produces similar reactions. Here, again, we are unable to see what benefit has been derived from the study of these last two considerations. All of these findings may, however, be considered acquisitions to our storehouse of knowledge, and as abstract facts may at some time, like the stone which was set at nought by the builders, become the head of a corner.

To return to our subject. It would seem that alienists would be more interested in this subject than internists, for to them it has not alone a scientific interest, but an eminently practical bearing. In this field, where conditions are so favorable and the work so promising (we refer to all gastric methods), it is remarkable that so little has been done, for, as von Noorden pointed out many years ago, scarcely any other physician is in so favorable a position to study his patients for months and years, under such excellent conditions for observation, as is the physician in the insane asylum.

Von Noorden, in 1887, was the first to conduct a series of experiments which had in view the determining of the effect of states of mental derangement upon the stomach secretions of the insane. The effect of mental attitudes upon certain gastrointestinal phenomena had long been observed in the medical clinics, and it was because of this that von Noorden sought to extend his observations to the field of insanity. He picked as the object of his research cases of melancholia, for the reason that in this mental state the gastric digestion is generally interfered with in the widest sense of the word. He eliminated all distinct diseases of the stomach, such as cancer,

ulcer, and dilatation, and restricted himself to a definite group of cases in the expectation of finding alongside of psychical fancies of a certain type symptoms of a corresponding type.

In his work von Noorden followed Riegel's (1887) scheme for the analysis of the stomach contents. This consisted in the estimation of the total acidity and the qualitative testing for free hydrochloric acid with tropæolin and Congo red paper, for lactic acid with Uffelmann's reagent, and for the peptic power by means of the egg-disk method. The fasting stomach was also examined, and the motor power determined by the amount recovered after an ordinary meal and the condition of the succeeding lavage.

Von Noorden reports in all 14 cases. The results of his investigations are as follows:

The first 12 cases represent states of pure psychical depression, differing from each other only in severity and duration. The first 10 of these correspond in all essential points, namely:

1. The evacuation of the stomach was hastened after the eating of the chief meal, consisting of food of a mixed character.

2. The total acidity reaches its highest percentage value during the process of digestion, as we do not find it in healthy men or only exceptionally so.

3. The strong acidity results entirely or almost entirely from the presence of free HCl, organic acids intrude seldom, and then only in small amounts.

4. The peptic power of the stomach is excellent in the presence of meat. The peptic power of the filtered stomach contents in the presence of the albumin disks is about normal.

5. A continual hypersecretion of the stomach fluids in the sense that at times when the stomach no longer contains food fluid secretion still continues, does not exist.

6. Dilatation of the stomach exists in no case.

Cases XI. and XII. showed what was considered normal secretion, and Cases XIII. and XIV., which had gone into dementia, showed hydrochloric acid secretion lower than normal.

Thus von Noorden overthrew completely the ideas previously held and revolutionized the treatment of stomach symptoms in melancholia, placing it upon a scientific basis. It is to be regretted that others have not extended the work farther and carried on investigations in other mental states.

Order of Our Investigation. For purpose of comparison, we have followed, with but few exceptions, the same order of investigation as did von Noorden. Where we made changes we did so because we thought that better and more constant conditions would be obtained.

The selection of patients was made by asylum physicians, with the approval of Dr. William M. Edwards, Medical Superintendent. Only those cases which showed signs of depression were selected,

and in all these we had sufficient grounds for making the diagnosis of melancholia. Many were in the beginning stages, while others were in a state of transition into secondary forms of insanity. In our routine work we found that many of the patients examined showed more or less severe stomach disease. These we rejected, retaining only those who showed no signs of organic disease, such as cancer, ulcer, and dilatation. We further endeavored to select patients who made no complaint of stomach symptoms. This was difficult to do, because of the great frequency of such symptoms in melancholia. Because of the great frequency with which we meet with enteroptosis among the insane as well as among patients in our internal medicine clinic, it was next to impossible to obtain sufficient women patients for our investigation without including a few of these (Case I. and Case II., Group II.). However, we do not hesitate to present these, for the reason that the stomach secretions in such cases are as likely to be normal as they are to be abnormal. The majority of such individuals have never had their attention drawn to their stomach until a hospital examination has discovered to them that some of their viscera are displaced.

The Test Meals. We chose as the principal test meal for our investigation the Ewald test breakfast or rather its equivalent: 70 gms. of white bread without crust and 300 c.c. of water. This was removed from the stomach one hour after eating. We selected this meal because it is in more general use among gastroenterologists and because we can better compare results with those obtained in the medical clinic. With but few exceptions we determined the effect of Riegel's test dinner and the ordinary asylum dinner upon the functions of the stomach. In this respect our work differs from that of von Noorden, who employed a mixed meal of soup, meat, potatoes, cabbage, turnips, carrots, spinach, etc., of which his patients were fed an amount that probably fell a little below what they would eat in their normal state. This meal was removed at the height of digestion, a time which corresponded in most of his cases to five or six hours after eating.

The majority of our patients took the food unwillingly after much coaxing, a few, however, seemed to eat with relish and manifested some interest in what we were doing. In no case did we have to resort to gavage.

Examination of the Fasting Stomach. This was done to exclude cases of hypersecretion and to assure us that in those cases where the stomach outlines by the distention test indicated a slight degree of dilatation we were in all probability dealing with a megastria and not with a pathological dilatation, and that in those cases where the stomach outline did not extend beyond what is considered normal, there was no pyloric obstruction of any kind.

Chemical Analysis. We employed for the chemical analysis of all stomach fluids Toepfer's method, controlling the hydrochloric

acid test with Gunzberg's reagent and Congo red paper. Lactic acid was tested for by Uffelmann's and Keeling's methods. We are thoroughly familiar with the objections raised to the employment of Toepfer's method, but this is no place to discuss methods; suffice it to say that it has many advantages over the method employed by von Noorden, and that the results obtained may be considered approximately correct. The titrating in each case was almost always made by the same person. This was done to avoid differences that might result from the personal equation. For well-known reasons we considered, however, as the end reaction for the total acidity the appearance of the first permanent pink[1] (not the deep red advised by Toepfer) for free hydrochloric acid, the disappearance of the orange tint.

The peptic value was estimated partly by the egg-disk method and partly by the Mett method. In the latter method the peptic value is expressed by the squares of the millimetres of egg white digested during ten hours' incubation and not by the number of millimetres, as is frequently but incorrectly done. The curdling ferment was tested qualitatively by Leo's method, and starch digestion by Lugol's solution.

Histories. We present a brief history of each case, together with the chemical analysis and other findings. Only those points are given which we think are sufficient to warrant us in deciding that the case is one of depression, as the object of our research is to inquire into the secretions of the stomach in these patients. *The physical examination may be considered negative excepting when otherwise indicated.* With few exceptions, urine, blood, and weight notes are given. These are taken from the asylum records.

Consideration of Cases. We report in all 22 cases and have arranged them into three groups. Group I. contains 16 cases, all of which have shown at some time degrees of hyperacidity. Group II. contains 2 cases, both of which show hyperacidity, but, because of certain findings, such as fragments of mucosa, it was thought best to consider them separately. Group III. contains 4 cases showing normal secretions and achlorhydria.

Group I.

In this group there are 8 females and 8 males as follows:

Case I.—Emma G. (see Table I.), aged sixty-one years, married, American, washerwoman, common school education; two children dead. Mother was insane latter years of her life. The patient had pneumonia nine years ago. Since this time her health has been very poor. She has overworked and has had much family trouble. Insanity began in the spring of 1899 with depression, gradually developing

[1] The standardizing of our decinormal soda solution was done by titrating to the same color.

into delusions of impending harm. Six months before entering the asylum she had an ovarian cyst removed.

The patient was admitted to Michigan Asylum for the Insane May 1, 1900. She was profoundly depressed. She would not speak and sighed almost constantly. She soon became agitated, moaned, and at times cried loudly. She was restless and frightened at everything. There has been little improvement during her residence at the asylum. She has suicidal tendencies, threatens to drown herself, but has made no suicidal attempts. She refuses to eat and has to be fed. She will not talk, except occasionally a few fragmentary words. She sits in one place all day with her eyes upon the floor. She escaped once from the asylum in her night-clothes. She has never complained of her stomach. Her appetite is poor and she does not sleep well.

Physical Examination. The patient is of slender frame, 154 cm. high. The musculature is small and wasted. The skin is sallow and anæmic. The tongue is pale, moist, swollen, and has a white coating all over. Her memory is impaired temporarily, indication is weak and mentalization is slow.

Heart: Soft blowing systolic murmur at apex, otherwise the heart is negative. Pulse, negative.

The abdomen is below the level of the ribs. There is slight bulging just below the umbilicus and a laparotomy scar. The abdomen is easy to palpate. There is no succussion in the stomach. On distending the stomach with air, the greater curvature is seen two and a half inches below the umbilicus, the lesser curvature at the umbilicus.

Pelvis: Slight laceration of cervix. Uterus is retroverted.

Blood: Red cells, 4,800,000; hæmoglobin, 72 per cent.; white cells, 3820.

Urine: Negative on all occasions.

The stomach secretions in this case were observed, as will be seen from the accompanying table, from May, 1900, to December, 1902. For eight months at the times when Ewald test meals were given, there was always, with but one exception, a distinct hyperchlorhydria.

CASE II.—Clara H. (see Table II.), aged sixty-eight years, married, American, housewife; three children; common school education. Her father was insane. The patient was always quiet and reserved in her manner until three months previous to her entrance to the asylum. At this time she began to be more talkative and apprehensive. She had delusions that her family were going to lose their property and that she was going to be killed; that she was possessed of a devil and that brimstone was coming out of her. She attempted suicide by cutting her throat.

The patient was admitted to the Michigan Asylum for the Insane in January, 1902. She was profoundly depressed and was sus-

picious of everything. As a rule, she would not talk. She worried much, and occasionally said that she was wicked. At times she became very restless and much agitated. Her appetite was irregular, sometimes very good; at other times she refused to eat. Her bowels were constipated. She did not complain of her stomach. May 24, 1902, she was less depressed than when she entered.

Physical Examination. The patient is fairly well nourished.

Abdomen: The abdomen is flat and below the level of the ribs. The panniculus is thin. The kidneys and spleen cannot be felt. Palpation is negative. It is almost impossible to find out more about her abdomen. She absolutely refuses and resists food and stomach tube. There is no evidence that her stomach is dilated or prolapsed.

Blood: Red cells, 4,000,000; hæmoglobin, 75 per cent.; white cells, 9772.

Urine: There is a trace of albumin, a few hyaline casts.

Weights: July 22, 1902, 83¾ pounds; May 31st, 94½ pounds; August 31st, 97½ pounds.

CASE III.—Lizzie W. (see Table III.), aged thirty-nine years, married, American, housewife. Family history negative. Syphilis suspected. Three months before admission to asylum she began to have delusions and was quite hypochondriacal. She was very irritable and at times became agitated. She thought her husband was going to murder her and she grew violent.

Patient was admitted to asylum February 9, 1901, in an extremely confused condition. She had delusions of persecution. Three days later she had one or two epileptiform attacks, later she grew noisy, and in the following July had a paretic stroke. She recovered from this and continued in her delusion that someone was trying to kill her. She refused to eat at times because, as she said, she knew the food contained poison. December 19th she was removed somewhat improved. January 2, 1902, she was returned in the same condition as before. She now had delusions that her clothes were filled with snakes. She was much depressed, crying much of the time. She died June 2, 1902, after a convulsion. No stomach complaints.

Abdomen, negative; stomach in normal position.

Gynecological Examination. Labia minora hypertrophied, laceration of cervix. Urine, negative.

Stomach observations were made at intervals from February, 1901, to July, 1902. During eleven months at the times when examinations were made a distinct hyperchlorhydria was found after the Ewald meal.

CASE IV.—Mary L. B. (see Table IV.), aged forty-eight years, single, American, housewife. One uncle insane. One aunt committed suicide. The patient has always been peculiar, easily excited, and easily irritated. In August, 1900, she began to show signs of insanity. She was much depressed. In February, 1901, she took

carbolic acid into her mouth, incurring some burns on her lips and tongue. She believed herself to be a great sinner and that she would never be saved. Her appetite was poor and she did not sleep well. She said her stomach never troubled her.

The patient was admitted to the Michigan Asylum for the Insane February 16, 1901. She was decidedly agitated and showed hysterical and suicidal tendencies. Her wickedness gave her much concern, and she would not be moved from the idea that she had ruined and disgraced her family. She showed suicidal tendencies. During her residence at the asylum her appetite improved and she slept better. Her mental condition, on the other hand, did not improve. She grew more restless and agitated, would not stay in bed and picked at her hands and face. She committed suicide by hanging.

Physical Examination. The patient is poorly nourished and looks somewhat emaciated.

Examination of Blood: Red cells, 4,000,000; hæmoglobin, 98 per cent.; white cells, 6663.

Urine: Negative.

Stools: Somewhat constipated.

Weight on admission, 103¼ pounds.

There has been practically no change in weight during her residence.

CASE V.—Hannah B. (see Table V.), aged fifty-seven years, married, American, housewife. One daughter insane. First signs of mental derangement began in October, 1899, supposedly due to worry over the death of her sister. She became melancholy, thought someone was watching her and was trying to take her away. She showed suicidal tendencies; indeed, she attempted to take her life by jumping into a well.

The patient was admitted to the Michigan Asylum for the Insane January 28, 1900. She is much depressed. She complains of her stomach being sour and a feeling of heaviness in the epigastrium. Her appetite is poor. During her residence at the asylum she steadily gained in physical and mental strength and was discharged January 1, 1901, cheerful and apparently free from depression. She was readmitted April 7, 1902, and at the same time her daughter was admitted. She remained quite well until recently, when she became irritable, obstinate, and emotional. She has worried over her daughter and is quite depressed. She attempted suicide by refusing to be removed from the railroad track. She continues to complain of her stomach. Her food comes up into her mouth; it is sour and produces a burning sensation in the tongue. She says that she has raised blood, bright red in color; it has a disagreeable taste and occurs in the morning and sometimes during the night. She does not know where it comes from. She vomits occasionally. She coughs considerably, but has not raised any sputum. She is constipated, and her appetite is poor.

Physical Examination. Patient is slightly emaciated, average-sized skeleton, skin is sallow; panniculus is thin; mucous membranes are pale. Tongue swollen, flabby, pale, coated white. Teeth are good.

Lungs: Thorax is long, broad, but shallow. Expansion is fair and symmetrical. Percussion is negative. On auscultation there are distinct crackling rales on the right side over the first and second ribs near the sternum; otherwise examination of the lungs is negative.

Heart: Apex inside nipple, fifth interspace costal. Heart dulness not increased. Heart sounds are weak. There is a very soft, faint, blowing, systolic murmur at the apex.

Abdomen: The abdomen is on a level with the ribs; the walls are very flabby; the panniculus is thin. There is tenderness on pressure over the epigastrium. Liver, spleen, and kidneys negative. Stomach negative.

Blood: Red cells, 4,000,000; hæmoglobin, 80 per cent.; white cells, 3552. Later red cells, 4,800,000; hæmoglobin, 77 per cent.; white cells, 8240.

Urine: No albumin, very few hyaline casts.

Weights: April 7, 1902, 131 pounds; June 30th, 131 pounds; July 31st, 134¼ pounds.

While there are some symptoms of ulcer in this case, our observation has led us to believe it does not exist, and that these symptoms are not sufficient to rule the case out.

CASE VI.—Olivia B. (see Table VI.), aged thirty-five years, single, American, dressmaker, common-school education. Mother is insane. The patient has always been well, of a kind and quiet disposition, fond of company and industrious. She has worked very hard and has eaten little. In March, 1902, she became insane; she had delusions that men on the street were pointing their fingers at her and making fun of her, that her customers did not care for her work any more, that she had done something wrong and that the people would not forgive her. She refused to eat because she thought the food contained poison.

The patient was admitted to the Michigan Asylum for the Insane April 7, 1902. She was depressed; she talked freely, however, and wondered why she was brought to the asylum. She is much confused and cannot reason; she keeps asking the same question over and over. She worries much over something she has done that cannot be forgiven. She is suspicious, cries much, and fears that something dreadful will be done to her. She has shown no suicidal tendencies as yet. Her sleep is disturbed. Her appetite is variable. She never complains of her stomach. Her bowels are constipated.

Physical Examination. The patient is well nourished and strong. Mucous membranes are slightly pale.

Heart: Apex fifth intercostal space, inside the nipple line. The heart dulness is not increased. There is a loud blowing systolic murmur at the apex transmitted into the axilla.

Blood: Red cells, 4,000,000; hæmoglobin, 75 per cent.; white cells, 5584.

Urine: Negative.

Weights: April 7, 1902, 109 pounds; May 31st, 109 pounds; August 31st, 111¼ pounds.

CASE VII.—Helen C. (see Table VII.), aged twenty-four years, single, American, schoolteacher. Paternal grandfather was insane, maternal grandmother died from some cord disease; father and mother both very nervous.

Patient is a college graduate, and was considered a bright student. She taught school one and a half years. Her career as a teacher was unsuccessful, and her work soon became distasteful to her. She became very nervous, and soon lost all control of herself. She would walk up and down her room, wring her hands, and talk in a confused manner.

Insomnia developed, and she became more depressed with the delusion that she had failed to seize opportunities for bettering herself and family, and for this reason they were coming to want.

Patient was admitted to the asylum February 25, 1902, in an extremely restless condition. She would run from one room to another, moaning and crying. She slept but little. Her appetite was variable. The following December she showed inclination to become violent by striking her fellow-patients. She left the asylum January 29, 1903. Since that time she was operated on for removal of the clitoris. She improved for a time, but is now quite nervous.

Physical examination at the time of her admission was negative, with the exception of a systolic murmur at the aortic and pulmonary areas and a somewhat anæmic appearance.

The abdomen is negative, stomach in normal position.

Gynecological Examination. Slight hypertrophy of labia minora and clitoris.

Blood: Red cells, 4,000,000; hæmoglobin, 58 per cent.; white cells, 1400.

Urine: Negative.

June, 1904, patient perfectly well and has been for some months.

CASE VIII.—Anna R. (see Table VIII.), aged fifty years, married, German, housewife. Admitted to asylum June 26, 1900. No insanity in family. About the time of the menopause (1898) she became depressed and agitated, worried much because she thought she had killed her son. Later on she became very incoherent and extremely restless; she walked the floor almost incessantly night and day, crying and moaning. She is very untidy in her general appearance and habits. She has hallucinations of hearing, delu-

sions of depression, ideas of persecution. Sensations are painful. Psychomotor processes decreased.

Her bowels are constipated; she complains of her food coming up in her mouth; she vomits occasionally. Her appetite at first voracious; later on became poor. She gained in weight at first, then lost. During her stay in the asylum, over one year, her depressive delusions did not abate; she constantly worried over imaginary troubles. She died December, 1901, after laparotomy (anterior fixation and perineorrhaphy).

Patient is fairly well nourished, and abdomen negative.

Blood: Red cells, 4,000,000; white cells, 4774; hæmoglobin, 85 per cent.

Urine: No albumin, but a slight excess of leukocytes; otherwise negative.

Weights: June 26, 1900, 95 pounds; June 30, 1901, 118½ pounds; November 30th, 128 pounds.

Postmortem was permitted, but only to ascertain condition of the pelvis. Stomach was not examined.

CASE IX.—Hugo A. (see Table IX.), aged thirty years, single, German, factory hand. Family and personal history unknown.

The patient's conduct until within ten days of his admission to the asylum was not observed to be particularly out of the ordinary. He was admitted October 10, 1902, when he became suddenly violent and maniacal. This mental state was very soon followed by depression and agitation. He became anxious about himself and worried much. He has delusions regarding his digestive organs, thinks they are displaced. He begs for an operation to replace them. His general appearance is that of depression. He becomes agitated at times. Has made no suicidal attempts. His appetite is good and his bowels normal. This state of depression continued over one month prior to our investigation of his case and still remains so.

Blood: Red cells, 5,240,000; white cells, 3424; hæmoglobin, 72 per cent.

Urine: Negative.

Weights: October 10, 1902, 144 pounds; 17th, 153 pounds; 24th, 149 pounds; December 4th, 144 pounds.

CASE X.—Frank W. C. (see Table X.), aged thirty-four years, married, American. Maternal uncle was insane. Patient had always been a man of good habits until his mental derangement began a few weeks before his admission. During this time he had been acting peculiarly, and on November 24, 1902, he developed the idea that people were following him for the purpose of killing him. He entered a hardware store for the purpose of purchasing a revolver, but was taken into custody.

Patient was admitted to the asylum November 27, 1902, with marked hallucinations of hearing. He was very suspicious of those

about him, fearful of injury, and thought he would be mutilated in some way before he would be allowed to leave the asylum.

He became very restless and confused mentally. He showed some signs of improvement and was discharged January 14, 1903. It was necessary to readmit him a few days later because of increasing delusions and hallucinations. He was much depressed, cried much, thought his wife was unfaithful. A few days later he again showed signs of considerable improvement and was discharged.

Physical examination shows apex beat slightly displaced to the left. There was slight roughening of the first sound at the apex.

Urine: Small amount of albumin, excessive leukocytes.

Blood: Red cells, 5,240,000; white cells, 6472.

CASE XI.—Hugh L. (see Table XI.), aged thirty-eight years, married, American. Family history negative. In May, 1902, patient developed peculiar ideas. Thought people were trying to injure his reputation and do him harm. He became restless and sleepless, and his appetite became capricious. The following November he attempted suicide twice. Tried to drown himself.

Patient was admitted to the asylum November 10, 1902. He thought he had done a great wrong to many people. That he had lost all his property and that his friends had deserted him. He was much depressed and felt that he was unfit to live. He refused food because he thought it was needed for someone else. In a few weeks he showed signs of improvement. His delusions grew less, and in the following May, 1903, he was discharged as recovered.

Patient was readmitted in October, 1903, having again become incoherent in his conversation and mentally confused. He was depressed and emotional. He showed improvement again, and was discharged February 6, 1904, practically improved.

Urine: Negative.

Blood: No report.

CASE XII.—George K. (see Table XII.), aged twenty-three years, single, American, teacher. No insanity in family. Personal history negative. In March, 1902, patient began to show signs of mental derangement. He became irritable and sleepless. He worried much about his bad habits, and thought his illness due to his meanness. He endeavored to avoid seeing people, preferring to be quiet. Thought people were watching him and would hide himself under the barn for hours. He was admitted to the asylum October 2, 1902, depressed and delusional. Believes he has been very wicked and that he is unworthy, begs to go home, so that he can ask forgiveness of everyone.

Abdomen just below the level of ribs, symmetrical. Palpation, etc., negative. On distending stomach with air, the greater curvature is found two fingers above the umbilicus.

Blood: Red cells, 6,000,000; hæmoglobin, 75 per cent.; white cells, 4569.

Urine: Negative.

Weights: October 1, 1902, 160 pounds; November 1st, 173 pounds; December 1st, 176 pounds.

CASE XIII.—Frank K. (see Table XIII.) aged thirty-eight years, married, Austrian, varnisher. Family and personal history unknown. June, 1902, he became depressed, moody, restless, and irritable. He was admitted to the asylum July 14, 1902, decidedly depressed, quiet, and unconcerned by what transpired about him. His sleep is poor; he has hallucinations of hearing and passes much of his time crying and sorrowing. His appetite is poor but he has no complaints concerning his digestion. His bowels are constipated.

Abdomen is on a level with the ribs. The stomach is in normal position.

Blood: Red cells, 4,300,000; haemoglobin, 85 per cent.; white cells, 2284.

Urine: Negative.

Weights: July 15, 1902, 143 pounds; 30th, 145 pounds; August 13th, 149 pounds; December 4th, 151 pounds.

CASE XIV.—Herman O. (see Table XIV.), aged twenty-four years, single, American, medical student. There is no history of insanity in the family. The patient has always been well and of more than ordinary mental ability. He showed no signs of mental derangement until December 3, 1900; at this date he purchased some arsenic and opium with the intention of committing suicide. For two years previous to this he worried much and complained of his stomach. It is thought that he applied himself too closely to his studies.

Patient was admitted to the asylum December 7, 1900. He was much depressed, introspective, and moody, and decidedly hypochondriacal. He has marked delusions concerning his stomach; he complains of distress in the stomach, but his complaints are vague and unsatisfactory. The bowels are normal.

Blood: Red cells, 3,000,000; haemoglobin, 90 per cent.; white cells, 5204.

Urine: Negative.

Weight: December 2, 1902, 154 pounds. We were unable to find other weights.

CASE XV.—George E. B. (see Table XV.), aged fifty-five years, married, American, farmer. Nothing known of family history. Patient had always been healthy until his insanity began in the spring of 1902. At this time he became greatly depressed, worried over small matters. He complained of his stomach, and it is thought by members of his family that when his stomach symptoms became aggravated his depression increased. About one month after the onset of his mental derangement he attempted to poison himself. His condition improved, however, in the course of a few months, but he soon returned to his state of depression, attempting to kill himself several times by hanging. His only delusion has been that

he considers it his duty to kill himself because of his unworthiness. At times he is agitated.

Urine: Negative.

Blood: Red cells, 4,860,000; white cells, 9265; hæmoglobin, 90 to 100 per cent.

Weights: December 10, 1902, 155 pounds; 17th, 157 pounds; 24th, 155 pounds; 31st, 155 pounds.

CASE XVI.—John V. (see Table XVI.), aged forty-five years, married, Hollander, common-school education. His mother was insane for a short time; otherwise family history is negative. Patient has always been a man of good habits. He was insane four weeks prior to his admission to the asylum, December 8, 1900. He began to act peculiarly, and for a time refused to speak; soon after this he became suddenly elated, yelling and whistling and making unusual disturbance. He did not sleep and talked incoherently. When admitted he was in a state of acute mania, which continued for four months, during which time he gained in weight and general bodily nutrition. In March, 1901, he began to be depressed. He sat around the ward worrying about himself and his family, and continued in this condition for eight months, when he was discharged as improved. When first deranged he had a voracious appetite, but later he became irregular about his eating.

Urine: Negative.

Blood examination was not made.

Weights: December 17, 1902, 146 pounds; January 10, 1903, 135 pounds; October, 146 pounds.

DISCUSSION. The results of the preceding examinations may be summed up as follows:

1. The evacuation of the stomach after the test breakfast is hastened in 1 case, normal in 6 cases, and somewhat delayed in 9 cases. After the Riegel or ordinary asylum meal the evacuation is hastened in 8 cases, normal in 1 case, and delayed in 3 cases. The remaining 4 cases received no large test meal. We may say in general that after a small stimulus, such as that received by the Ewald test breakfast, the evacuation of the stomach is usually normal or somewhat hastened; while after the large stimulus of the Riegel and ordinary asylum meal it is more frequently hastened.

2. The free hydrochloric acid value ranges from 60 to 90 per cent. in 12 cases, from 50 to 58 per cent. in 4 cases. These may all be justly called cases of hyperchlorhydria.

3. The total acidity ranges from 90 to 111 per cent. in 10 cases, from 73 to 88 per cent. in 6 cases. It is always above what is usually accepted as normal. They may all be classed as cases of hyperacidity, according to the method of Riegel as employed in 1887, and they accordingly correspond to those reported by von Noorden.

4. The peptic value is up to normal in all cases, often somewhat

increased. The latter condition is especially noticeable in those cases where Mett's method was employed.

5. Hypersecretion is present in no cases.

6. Lactic acid and signs of fermentation are never encountered.

7. The hyperacidity may be said to be constant in most cases, as it persisted for a few months, and in a few cases for many months. The question of the constancy of the secretions was raised by von Noorden, as his cases were observed only over a short period.

8. Retention and dilatation exist in no case. Case I. shows a distinct gastroptosis.

9. Fragments of mucosa were found only in 1 case; unfortunately this was lost and its character is not known. It was a small superficial piece.

10. Stomach symptoms were complained of in 4 cases, Cases V., VIII., XIV., and XV. In Case V. the symptoms were those of hyperacidity.

11. Mucus was not excessive in 13 cases. Slight excess was noted in 3.

12. Males and females equally affected. Von Noorden's observations were upon women only.

EXPLANATIONS. (Ad. I.) It is impossible to fix a definite line for the normal amount of residue after any meal. This has to be left to the judgment of the practised eye. If we find only a few c.c. on an empty stomach three or four hours after an ordinary dinner or a Riegel meal, we say that the evacuation is increased. If we find 50 or 60 c.c., or possibly a little more, we say that the evacuation is about normal. If, on the other hand, we find that 100 c.c. or over are present, we say that the evacuation is delayed. The amount is estimated by what is recovered before and after the lavage. A somewhat extended experience in the University Hospital and in private practice had led to the belief that the majority of so-called normal stomachs contain some food as long as five hours after the ordinary mid-day meal, which is the heavy meal of the day in this locality.

(Ad. II.) The question of normal acidity values is also a perplexing one. The stomach is capable of very wide daily variations. But if we take, for instance, the average figures of Riegel for normal free HCl secretion, 20 to 40 per cent., and consider 50 to 70 per cent. moderate increased secretion, and 71 to 100 per cent. high secretion, then the large majority of our cases are confined to the middle group. Von Noorden did not touch upon this point, as he made no direct estimations of free HCl.

(Ad. III.) We may here compare our results with those of von Noorden, and to do this we must use his figures. He arranged three groups of acidity values; from 0.24 to 0.29 per cent., or, according to the Ewald notation, from 66 to 80 per cent. slight secretion. From 0.30 to 0.39 per cent., or, according to the Ewald notation, 80 to 100

per cent., medium secretion. From 0.40 to 0.50 per cent., or, according to the Ewald notation, 100 to 115 per cent. and higher, highest degrees of secretion. The majority of our cases occupy the middle group; they rarely intrude upon the third group, but in a few instances are found to occupy a position in the upper part of the first group. It will be seen that the variations in some cases are quite great. In all of these respects our observations are remarkably parallel with those of von Noorden, with the exception that our stimulus was less. The total acidity is in all cases above the generally accepted 70 per cent. as the beginning of hyperacidity after an Ewald meal.

(Ad. IV. to X.) These points require no further explanation than will be found under "Order of Investigation."

(Ad. XI.) The amount of mucus was estimated by inspection with the aid of a glass rod or the fingers. The saliva was prevented as far as possible from getting into the stomach contents. Excess of mucus was noted when ropy strings were present.

Group II.

Case I.—Frank S. (see Table XVII.), aged fifty years, single, American, cabinetmaker. One sister mentally deranged. No other history of insanity in family. Patient has always had good health. He was of a cheerful temperament, led a moral life, and was above the average in mental ability. He was of an inventive turn of mind, and his mental derangement is attributed to disappointment over the failure of some of his inventions. Insanity first manifested itself by the gradual oncoming of depression and melancholy. He became sleepless. His appetite became poor, and his general health began to fail. He complained of constipation and loss of weight.

Admitted to the asylum January 3, 1901, six months after the first signs of mental change. He was very quiet, downcast, and spoke only in a whisper. He had delusions which were mostly somatic. He made no definite complaints concerning his stomach, except that at times there was a feeling of distention. He attributed his condition, however, to something wrong with his bowels and insisted that this could never be improved. He had a delusion that his stomach was made of some hard material, that his food was not digested, and that nothing ever passed his bowels. He manifested suicidal tendencies, professing that if he "had the nerve" he would kill himself. Attempted suicide once by hanging. His bowels are constipated. He constantly worries.

Patient is somewhat undernourished.

The abdominal walls are flabby. The panniculus is thick. When distended with air the greater curvature of the stomach is seen to be just at the umbilicus. Otherwise the abdomen is negative.

Blood: Red cells, 4,800,000; hæmoglobin, 75 per cent.; white cells, 4061.

Urine: Negative.

Weights: July 1, 1902, 164 pounds; May 28th, 168 pounds; July 23d, 165 pounds; September 11th, 157 pounds.

This patient was under observation for fifteen months. Hyperchlorhydria was present practically during all this period.

Dr. Warthin's report on the fragment of mucosa found in the eye of the stomach tube April 20, 1901.

Portion of gastric mucosa cut obliquely; entire mucosa atrophic; excessive mucous formation; majority of gland cells show some mucin; few acid cells; no lymphoid tissue; slight increase of interstitial tissue; slight small-celled infiltration of the upper portion of the mucosa.

Pathological Diagnosis. Early stage of atrophic catarrhal gastritis. (See Fig. 1.)

FIG. 1



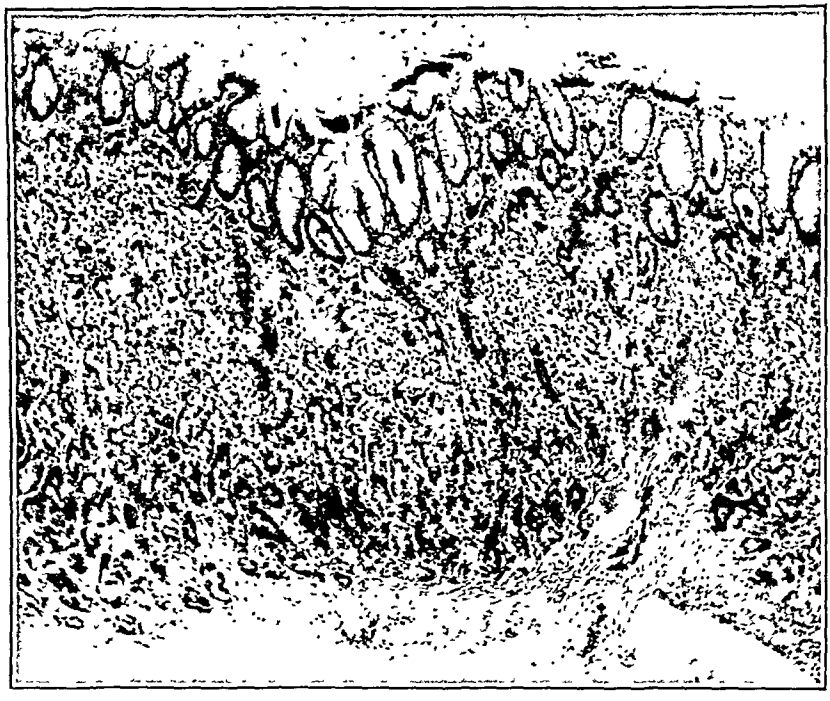
Frank S. Section from bit of mucosa found, April 20, 1901.

CASE II.—Martha Y. (see Table XVIII.), aged fifty-six years, married, American, housewife. Patient has three children and has been above the average in mental ability. One brother committed suicide; otherwise family history is negative. Insanity began three weeks before admission to the asylum in September, 1899. It was thought to be due to the menopause. She began to write letters to

various people stating that she had sinned against the Holy Ghost and that her wickedness could never be forgiven. She imagined that she had ruined her family and that she robbed her husband of $75,000. She had hallucinations of impending harm to herself and children; also hallucinations of taste and hearing. It was not long before she became somewhat agitated. She is extremely depressed.

After eight months' stay in the asylum she had improved slightly, was discharged, but was returned in a few weeks in the same condition as before. She has threatened suicide frequently. She has

FIG. 2



Martha Y. Section from bit of mucosa found, February 26, 1901.

had a delusion that her bowels have not moved for six months. June 1901, attempted hanging. She constantly brooded over her wickedness.

Patient's appetite has always been good; she constantly laments because she feels inclined to eat too much, but at no time has she complained of symptoms referable to her stomach. Says that she has no stomach.

Patient is well developed. Panniculus is quite thick. The abdomen is level with the ribs, walls are firm, making palpation difficult. No tenderness at any point. Examination is negative.

Stomach: The lesser curvature is one and a half fingers above the umbilicus, greater curvature two and a half to three fingers below.

Gynecological examination shows cystocele, rectocele, slight perineal laceration.

Blood: Red cells, 3,550,000; white cells, 4800; hæmoglobin, 75 per cent.

Urine: Negative.

Note August 5, 1902. Patient has deteriorated in physical health. When she entered the asylum she weighed 152 pounds, now 99 pounds. Since her admission she has had an attack of diarrhœa, and during the epidemic of smallpox in the institution in 1901 she was one of the most severely attacked. These two conditions are responsible for her loss in weight. There is no evidence of malignant disease.

Patient died after laparotomy. No autopsy could be obtained. The abdomen was, however, opened and the stomach was found to be of normal size and free from any new-growth or ulceration. The patient had been under observation for eighteen months, during fifteen of which hyperchlorhydria was always present after an Ewald meal.

Dr. Warthin's report. Fragment found February 27, 1900.

Bit of mucosa extending in places to the basement membrane, portions of which are present; mucosa is atrophic, less than two-thirds normal thickness; no lymph follicles present; very few acid cells; capillaries conjested; no increase of interstitial tissue; no excessive mucus formation.

Diagnosis: Simple atrophy. (See Fig. 2.)

Discussion. A careful consideration of both of the cases in this group shows that, aside from the finding of fragments of mucous membrane in the stomach contents, they correspond exactly with those of the first group. These bits of mucosa were placed almost immediately into 95 per cent. alcohol, and because of the appearance of fresh blood, their shape, and relation to the tube, there can be no question but that they were torn from the mucosa during the process of emptying the stomach and the withdrawal of the tube. They may therefore be looked upon as representing the true conditions going on in the area of the mucosa from which they came, uninfluenced by any action of the gastric juice after detachment from their blood supply.

In both cases there is evidence of atrophic change. In Case I. the atrophy is more noticeably associated with a progressive gastritis or degeneration; while in Case II. there is no excessive mucous formation, but the change is distinctly one of simple degeneration or atrophy of the glandular elements, resulting in a thinning of the entire mucosa. It is true that we are here dealing with a very limited portion of the mucosa only, and that we can make no positive statement of what is taking place in the mucosa beyond this limited area.

However, there is every reason to believe that the changes are at least going on in a more or less circumscribed area larger than the fragments found in the washings, for in the latter case pieces of mucosa were found on three different occasions, the last piece being found three months after the second, and in all similar changes are seen. Further, the pathological change is more or less evenly distributed throughout the sections. It may, therefore, be assumed that there are other areas of the same nature scattered throughout the mucosa. This being the case, there would be reason to believe that with such a change, resulting in compression and degeneration of the glandular elements, the secretions, under ordinary circumstances, would be lessened. The question that naturally follows is, What is the cause of the apparent oversecretion or hyperacidity?

In answer to this question, three theories may be advanced:

1. That at some time the patients have had a much higher hydrochloric acid secretion and that the present more or less constant secretion is due to progressive anatomical changes.

2. That the remaining intact glands are doing compensatory service.

3. That the increased secretion is due to the influence of the neurosis or psychosis.

Knowing that the stomach glands are capable under normal circumstances of performing unlimited service according to the demands made upon them, as is shown, for instance, in the greater secretion after an albuminous meal than after a meal made up principally of carbohydrates, and in view of the fact demonstrated by von Noorden and ourselves that hyperacidity is present in a large percentage of cases of mental depression, we are inclined to the third opinion and think it is not unreasonable to expect that, in spite of the progressive anatomical change, the neurosis or psychosis is responsible for the increased secretion.[1]

Group III.

This group contains the remaining cases of our series. Cases that were selected in the same way as those that have gone before. They do not meet the requirements set by von Noorden in order to be considered cases of hyperacidity. Cases I. and II. have, however, fairly high hydrochloric acid values at times. Cases III. and IV. are practically cases of the so-called achylia.

Case I.—Arthur P. (see Table XIX.), aged forty-three years, married, American. Little known of family history, except that he

[1] Hemmeter reports twelve cases of anacidity and subacidity in which fragments of mucosa were found Proliferation of the glands was present in one case with well-marked round-celled infiltration.

was always weak-minded, a bar-room lounger, and a rough fellow in general. Six months prior to his admission to the asylum he threatened to assault his father with an axe. He wandered from place to place.

Patient was admitted to the asylum March 1, 1893. He was easy to manage, observed the rules of the institution well and had no idea of his mental condition. The following June he developed fault-finding and whining, frequently showing a mean disposition, annoying other patients. March 19, 1894, showed much excitement; thought he was going to die; that his heart was moved from its normal place and that it was bleeding. He became confused and thought that there was something inside of him that migrated with regularity from his breast bone to his rectum. September 22, 1896, these hallucinations were still present, and he was if anything more depresed than before. In March, 1901, complained of various animals in his abdomen; thought someone was trying to poison him. He now thinks that the lining of his stomach is loose and turned upside down. He eats well and is much improved in physical strength.

Physical examination is entirely negative.

CASE II.—Wilhelmina D. (see Table XX.), aged sixty years, married, housewife, native of Holland. One brother insane. Patient has always been of a nervous temperament; her mental ability was good. At the age of fifty she became insane, but recovered from this attack in about thirteen weeks. In April, 1897, she became insane again; she imagined that she was lost forever; that she had cancer of the stomach. She insisted that she felt something crawling in her stomach. She was frequently nauseated and at times vomited. Her sleep was disturbed and her appetite was poor.

The patient was admitted to the Michigan Asylum for the Insane September 28, 1897. She was depressed and worried because she thought she had a cancer. She abandoned this idea quite a little, however, when she was assured that such was not the case. Her delusions were those of hypochondria; she had a pain in a new place every day. She was removed from the asylum November 10, 1897; readmitted December 28, 1902, after having been in several institutions and taking a sea voyage. She was no better and had very painful delusions. She thinks her husband is going to shoot her, that people are running electric wires into her house, and that someone is going to chloroform her. She complains of her bowels rumbling constantly, so that she cannot rest; she is sure this condition is worse when the stomach is empty. She is much given to whining. It is thought that she is a morphine-eater. She has improved quite a little since her readmittance. Her appetite is good. She sleeps better, but her bowels are somewhat constipated.

Abdomen: There is tenderness in the region of the liver.

TABLE I.—Emma G.

Explanation of characters: + ++ = Lugol. M.B., V., R.V., R., and A.C. = Experimental meal.

Number.	Date.	Meal employed.	Motility tests. Time in stomach.	Amount recovered.	Condition of stomach washings after removing test meal.	Acids. Lactic acid (Uffelmann).	Hydrochloric acid (Gunzberg).	Free hydrochloric acid.	Combined hydrochloric acid.	Total acid.	Ferments, etc. Pepsin egg tablet digested in	Rennen coagulation of milk occurred in	Starch digestion (Lugol).	Bits of mucosa.	Important micro-organisms.	Remarks.
	1900		hours	c.c.							hours	min.				
1	May 20	Ewald	1	21	Clear	0	++	36	42	111	3½	...	...	...	...	
2	" 20	Riegel	4	0	Much meat	...	...	...	...	...	...	...	...	...	...	By mistake meat was not chopped; tube clogged.
3	Dec. 6	Ewald	1	110	Practically clean	...	++	57	32	98	4	8	M.B.	0	0	
	1902															
4	Jan. 3	"	1	45	Very little bread	0	++	72	4	94	4	5	V.	0	0	Alizarin titration made with 1 c.c.
5	" 4	"	1	40		0	++	64	8	78	...	5	V.	0	0	Patient hard to manage; no lavage given.
6	" 5	"	1	35	Much bread	...	++	65	12	82	4	...	R.V.	0	0	Titrations made with 1 c.c. except for total acid.
7	" 14	Asylum supper	14	0	See remarks	...	...	...	...	...	...	...	...	0	0	Only a few blood-stained strings of mucus could be obtained by washing.
8	" 18	Riegel	3½	0	Many small meat bits	...	0	...	...	...	...	...	...	...	...	A number of small meat bits found in wash-water.
9	Dec. 2	Asylum breakfast	4	...		...	...	35	10	50	...	...	...	...	...	

TABLE II.—Clara H.

	1902		hours	c.c.							hours	min.				
1	Feb. 20	Ewald	1	80	Practically clean	0	+	78	20	108	4⅓	...	M.B.	0	0	
2	" 26	"	1	45	Practically clean	0	+	78	20	108	5	...	...	0	0	
3	" 28	Asylum supper	15	0	Nothing	...	...	...	...	...	...	...	...	...	...	Fasting stomach; patient absolutely refuses to eat any more test meals.

TABLE III.—Lizzie W.

			hours	c.c.				hours min.							
1	1901 Feb. 22	Ewald	1	80		0	+	61	18	92	3 10	R.	0	0	Lavage could not be given; patient much agitated.
2	1902 Jan. 27	"	1	150		0	+	50	18	84	... 10	R.V.	0	0	Very little mucus in washings.
3	" 31	"	1	80		0	+	62	28	96		R.	0	...	Stomach fluid stood 24 hrs. before examined; no evaporation.
4	Feb. 1	"	1	125		0	+	18	20	76		R.	0	0	
5	Nov. 30	"	1	65		0	+	12	1	11	36 x	R.V	...	...	
6	Dec. 2	Riegel	3	50	Some food	0	+	25	30	65	mm.*	...	...	...	
7	" 3	Asylum supper	11	3	Little mucus	...	...	...	...	...		...	...	...	Fasting stomach.

* Peptic value, Mett's method.

TABLE IV.—Mary B.

			hours	c.c.				hours min.							
1	1901 Feb. 22	Ewald	1	80		0	−	60	26	92	1 5	R.V.	0	0	Mucus not excessive.
2	Mar. 4	"	1	60		0	+	42	12	72	2¼ 5	R.V.	0	0	Considerable mucus.
3	" 5	"	1	55		...	+	54	18	76	1¾ 5	R.V.	0	0	
4	" 6	"	1	80		0	+	46	32	88		R	0	0	

TABLE V.—Hannah B.

			hours	c.c.				hours min.							
1	1902 April 11	Ewald	1	60		0	++	62	10	78		R.V.	0	..	Patient difficult to manage.
2	" 12	"	1	80		0	++	52	6	66		R.V	0	x	Few yeast cells and bacteria.
3	" 13	"	1	60		0	++	48	10	61	. ..	R.V	0	x	Many yeasts.
4	May 20	Riegel	4	few		...	...	..	...	acid		...	0	...	Patient cannot be managed.

TABLE VI.—Olivia B.

Number.	Date.	Meal employed.	Motility tests. Time in stomach.	Amount recovered.	Condition of stomach washings after removing test meal	Acids. Lactic acid (Uffelmann).	Hydrochloric acid (Gunzberg).	Free hydrochloric acid.	Combined hydrochloric acid	Total acid.	Ferments, etc. Pepsin egg tablet digested in	Rennin coagulation of milk occurred in	Starch digestion (Lugol).	Bits of mucosa.	Important micro-organisms.	Remarks.
	1902		hours	c.c.							hours	min.				
1	May 11	Ewald	1	250		0	++	46	20	74	4	...	M.B	0	0	Lavage omitted; patient violent.
2	" 16	"	1	200		0	++	52	14	72	4	...	M.B.	0	0	" " " "
3	" 23	Riegel	3½	150		...	++	48	30	96	3½	0	A.C.	0	0	Milk not curdled after two hours in oven; chymosinogen test was not made.
4	July 19	Ewald	1	200		0	++	42	12	71	4	...	A.C.	0	0	Digestion of egg tablet complete.
5	" 19	Asylum supper	14	0												

TABLE VII.—Helen G.

	1902		hours	c.c.							mm.*	min.				
1	Dec. 30	Ewald	1	170	Clear	...	++	48	12	67	11	+	M.B	0	0	
2	" 31	"	75 m.	58	"	...	++	50	16	73	15.5	+	R.V.	0	0	
	1903															
3	Jan. 1	"	90 m.	60	"	...	++	50	13	70	15	+	R.V.	0	0	Fasting stomach completely empty.
4	" 3	"	50 m.	...		...	++	30	14	68	7.8	+	...	+	0	Last observation of experimental meal.

* Peptic value, Mett's method.

TABLE VIII.—Anna R.

	1900		hours	c.c.							hours	min.				
1	Dec 1	Ewald	1	65	Clear.	0	++	52	26	84	6	7	R.V.	0	0	
2	" 1	Asylum dinner	3½	...	"	0	0	...	...	...	...	...	...	...	...	Only a few shreds of meat recovered.
3		Asylum supper	15	...	"	0	0	..	...	acid	...	...	...	...	...	Fasting stomach; very small amount of mucus recovered.

TABLE IX.—Hugo A.

	1902		hours	c.c.							mm.*					
1	Nov. 30	Ewald	1	57	Clear	0	++	31	20	74	11	+	R.V.	0	0	
2	Dec. 1	"	1	112	Little food	0	++	10	1	64	9½	+	"	0	0	
3	" 1	Asylum dinner	3½	5	" "	0	++	20	...	11	9	+	"	0	0	
4	" 2	Ewald	1	30	Much food	0	++	30	7	57	7.5	+	"	0	0	
5	" 2	Riegel	4	40	Clear	0	++	17	20	60	1	+	"	0	0	
6	" 23	Ewald	1	70	"	0	++	77	12	102	8.5	+	"	0	0	
7	" 24	"	1	36	"	0	++	60	11	81	10.5	+	"	0	0	
8	" 30	"	1	180	"	0	++	67	15	92	10.5	+	"	0	0	
9	Jan. 2	"	1	...		0	++	30	2	10	8	+	"	0	0	Last observation of experimental meal.
10	Dec. 1	Asylum supper	14	15	Few bits toast	...	...	...	...	...	...	...	...	...	...	Fasting stomach; patient was seen eating toast a short time before.

TABLE X.—F. W. C.

	1902		hours	c.c.							mm.*					
1	Nov. 30	Ewald	1	68	Clear	0	++	60	16	90	5	+	R.V.	0	0	
2	Dec. 1	"	1	70	"	0	++	54	26	100	11.5	+	"	0	0	
3	" 1	Asylum dinner	3½	40	Much food	0	++	70	20	100	12.5	+	"	0	0	
4	" 2	Ewald	1	55	Little food	0	++	65	12	90	12.5	+	"	0	0	
5	" 2	Riegel	4	25	Clear	0	++	55	15	75	16	+	"	0	0	
6	" 23	Ewald	1	42	"	...	++	60	10	79	10.5	+	"	0	0	
7	" 24	"	1	28	"	...	++	57	8	76	11	+	"	0	0	
8	" 26	"	70 m.	30	"	...	++	54	7	67	8	+	"	0	0	
9	" 28	"	45 m.	0	"	...	+	35	...	48	8	+	"	0	0	Last observation of experimental meal.
10	" 1	Asylum supper	14 h.	0		...	...	...	...	...	...	...	...	0	0	

TABLE XI.—Hugh L.

	1902		hours	c.c.							mm.*					
1	Nov. 30	Ewald	1	208	Clear	0	++	50	0	70	3	+	R.V.	0	0	
2	Dec. 1	"	1	205	"	0	++	54	0	71	11.75	+	"	0	0	
3	" 1	Asylum dinner	3½	60	Little food	0	++	31	16	70	8.5	+	"	0	0	
4	" 2	Ewald	1	114	Much food	0	++	60	5	75	12.33	+	"	0	0	
5	" 2	Riegel	4	118	Clear	0	++	68	19	98	14	+	A.C.	0	0	Stomach washings before meal perfectly clear.
6	" 23	"	1	350	"	...	++	56	6	70	11	+	R.V.	0	0	
7	" 24	"	1	150	"	...	++	50	8	65	9.5	+	"	0	0	
8	" 26	"	70 m.	150	"	...	++	43	10	55	12	+	"	0	0	
9	" 28	"	1 h.			...	++	40	5	56	...	+	"	0	0	Last observation of experimental meal.
10	" 1	Asylum supper	15	0		...	...	...	...	...	...	...	...	...	...	Fasting stomach.

* Peptic value, Mett's method.

TABLE XII.—Geo. K.

Number.	Date.	Meal employed.	Motility tests: Time in stomach.	Motility tests: Amount recovered.	Motility tests: Condition of stomach washings after removing test meal.	Acids: Lactic acid (Uffelmann).	Acids: Hydrochloric acid (Gunzberg).	Acids: Free hydrochloric acid.	Acids: Combined hydrochloric acid.	Acids: Total acid.	Ferments, etc.: Pepsin egg tablet digested in	Ferments, etc.: Rennin coagulation of milk occurred in	Ferments, etc.: Starch digestion (Lugol).	Ferments, etc.: Bits of mucosa.	Ferments, etc.: Important micro-organisms.	Remarks.
	1902		hours	c.c.							mm.*					
1	Dec. 30	Ewald	1	156	Clear	0	++	40	20	80	7	+	R.V.	0	0	
2	Jan. 1	"	1	95	"	0	++	38	19	70	13	+	"	+	0	Suspected tissue bit found, but was accidentally lost.
3	" 1	Asylum dinner	3½	76	Little food	0	+	16	8	32	2	−	"	0	0	Mucus slightly increased.
4	" 2	Ewald	1	90	Clear	0	++	51	7	65	16	+	"	0	0	
5	" 2	Riegel	4	32	"	0	++	90	10	108	14	+	A.C.	0	0	Stomach washings before meal were perfectly clear.
6	" 23	Ewald	1	38	"	...	0	0	2	12	8	+	R.V.	...	...	
7	" 24	"	1	30	"	...	0	0	0	10	6	+	"	...	...	
8	" 26	"	1	52	"	0	0	0	0	12	7	+	"	...	...	
9	Feb. 1	Asylum supper	14	0		...	...	...	...	...	...	...	...	..	...	Fasting stomach.

TABLE XIII.—Frank K.

	1902		hours	c.c.							mm*.					
1	Nov. 30	Ewald	1	35	Clear	0	++	30	14	64	11	+	R.V.	0	0	
2	Dec. 1	"	1	70	"	0	++	27	17	50	12	+	"	0	0	Mucus slightly increased.
3	" 1	Asylum dinner	3½	5	"	0	...	...	...	...	...	...	...	0	0	
4	" 2	Ewald	1	90	Little food	0	−	8	7	27	⅔	+	A.C.	0	0	
5	" 2	Riegel	4	10	Clear	0	+	10	5	25	0	+	M.B.	0	0	
6	" 23	Ewald	1	155	"	...	++	50	11	68	12	+	R.V.	0	0	
7	" 24	"	1	60	"	...	++	71	10	92	9 5	+	"	0	0	
8	" 26	"	1	100	"	...	++	50	14	70	11	+	"	0	0	
9	" 29	"	1	..		...	+	52	10	78	8	+	"	0	0	Last observation of experimental meal.
10	" 1	Asylum supper	0	...		...	...	...	...	...	...	...	...	...	...	Fasting stomach.

* Peptic value, Mett's method.

TABLE XIV.—Hermon O.

			hours	c.c.							hours	min.				
	1900															
1	Dec. 8	Ewald	1	90	Some bread	0		28	12	50	5	10	M.B.	0	0	
	1901															
2	Mar. 5	"	1	150		0	+	50	18	78	5	10	R.V.	0	0	
3	" 6	"	1	170		0	+	62	18	88	...	...	"	0	0	
	1902															
4	Dec. 1	Asylum breakfast	3.5	50	Clear	0	+	30	6	64	5	½	"	0	0	Bread, butter, potatoes, coffee.
5	" 7	"	2	98	Much bread	+	0	0	15	10	0	½	"	0	0	Hash, gravy, oatmeal, bread, coffee.
6	" 7	Fasting	14	0		...	...	...	...	...	...	...	...	0	0	

TABLE XV.—Geo. E. B.

			hours	c.c.						mm.*						
	1902															
1	Dec. 31	Ewald	1	80	Clear	...	++	62	15	87	11	"	R.V.	...	...	
	1903															
2	Jan. 1	"	1.5	110	"	...	+++	63	11	86	12	"	"	...	...	
3	" 2	"	1	21	"	...	+++	62	5	85	12½	"	"	...	...	Last observation of experimental test meal.
4	" 3	"	1	...		...	++	62	18	87	...	"	...	...	...	Fasting stomach.
5	" 3	Asylum supper	15	0	Clear	...	-	...	...	...	...	...	...	...	...	

* Peptic value, Mett's method

TABLE XVI.—John V.

			hours	c.c.							hours	min.				
	1901															
1	April 19	Ewald	1	65		0	+	56	18	80	0½	...	R.V.	0	0	Patient became cyanotic; tube withdrawn early; no lavage.
2	" 20	"	1	20		...	+	58	16	78	...	10	"	0	0	Patient cyanotic; crying.
3	" 22	"	1	50		0	+	58	22	70	0½	8	B.	0	0	" " "
4	" 25	Asylum supper	15	0		..	...	...	...	...	...	...	"	0	..	Fasting stomach.
5	" 26	Asylum dinner	4	0	Much food	...	...	...	...	...	...	...	"	...	...	Hard to manage.

TABLE XVII.—Frank S.

Number.	Motility tests. Date.	Meal employed.	Time in stomach.	Amount recovered.	Condition of stomach washings after removing test meal.	Acids. Lactic acid (Uffelmann).	Hydrochloric acid (Gunzberg).	Free hydrochloric acid.	Combined hydrochloric acid.	Total acid.	Ferments, etc. Pepsin egg tablet digested in	Rennin coagulation of milk occurred in	Starch digestion (Lugol).	Bits of mucosa.	Important micro-organisms.	Remarks.
	1901		hours	c.c.							hours	min				
1	Apr. 19	Ewald	1	8		0	++	..	...	...	4	...	R.V.	0	0	Color dark brown; some altered and fresh blood.
2	" 20	"	1¼	1	Few shreds of mucus.	...	...	...	...	...	...	...	...	+	0	Nothing could be obtained after 15 minutes' effort; tissue bit in eye of tube
3	" 22	"	¼	20	"	0	++	40	30	80	3	8	R.V.	0	0	Color brownish; many R.B.Cs.; 1 c.c. used for each test.
4	" 24	Leube	2¾	0	"	..	...	...	...	...	...	...	...	0	...	
	1902															
5	Jan. 6	Ewald	1	0	Few flakes of bread	...	...	...	...	acid	...	...	...	0	...	
6	" 7	"	1	55	Clean	0	++	64	10	90	3½	10	R.V	0	0	Few fat globules from supper the evening before.
7	" 8	"	1	3		...	faint	...	...	...	...	...	...	0	...	Only a few c c. of brownish mucus could be obtained
8	" 10	"	1	4	Much bread	...	"	..	...	...	...	...	...	0	...	Nothing could be obtained after 15 minutes' effort until lavage was given.
9	" 11	"	1	75	Practically clean	0	++	46	20	72	3½	...	R.	0	0	
10	" 12	"	1	15	Much bread	...	++	...	...	...	...	...	...	0	...	
11	" 13	Asylum supper	15	0	Clean	...	...	...	...	...	...	...	...	0	...	No test for HCl could be obtained in wash water.
12	" 14	Asylum dinner	2¾	few		...	...	...	...	...	...	...	...	0	...	
13	" 18	Riegel	3½	"	Clean	...	...	...	...	...	...	...	...	0	...	
14	July 11	Ewald	1	150		0	++	56	28	90	4	...	R.V.	0	...	
15	" 19	"	1	100		0	++	51	8	68	+	...	...	0	...	

TABLE XVIII.—Martha Y.

				hours	c.c.							hours	min.				
1	1900 Nov.	6	Ewald	1	20	Clear	...	+ +	10	11	104	5	...	...	0	0	Ate very little milk before test meal.
2	1901 Feb.	26	"	1	21		0	+ +	50	20	104	2½	10	M.B.	+	0	One small bit mucous in eye of tube.
3	"	27	"	1	65	Clear	0	+ +	36	12	72	3	.	R.V.		0	One large bit mucous.
4	Dec.	10	Asylum dinner	3½	0	Small bit carrot	...	...	...	..	...	..	.	..	...	...	Meal of meat and potatoes, carrots, bread pudding; only few flecks of mucus.
5	"	17	Ewald	1	35	Clear	0	+ +	15	15	75	..		...	..	..	Titrations made with 1 c.c.
6	"	18	"	1	30	"	0	+ +	40	10	70	3½	10	R.V.	.	0	One small bit of mucous.
7	"	20	"	1	40	"	..	. .	70	30	100	1		"	0	0	Contents kept over night; from no stagnation; two titrations.
8	1902 Jan.	3	"	¾	35		0	+ +	11	6	66	1	5	"	"	0	
9	"	4	"	1	20	Clear	0	+ +	60	10	70	..	5	"	0	0	Test made with 1 c.c.; repeated.
10	"	5	"	1	15	Few flecks mucus and bread	...	+	18	10	62	3½	...	"	0	"	
11	Feb	19	Asylum dinner	2¼	40	Much	x	0	20	35	90	..		.	"	0	Meal of beef, potatoes, bread, milk, cake; very faint reaction with Ehrlich's.
12	Jan.	12	Asylum supper	11	0	Few flecks mucus	..		...	...	...	..		.	"		
13	"	14	Asylum dinner	3	much		...	0	...	...	...	..	...	.	..	.	
14	May	2	Regel	3.5	20	Few bits meat	0	+ +	31	...	60	...		..	...	...	
15	"	22	Asylum dinner	3	0	"	...	...	...	...	..	...	...	..	...	...	Meal of mac. soup, beef, bread, butter, milk, onions. Fasting stomach examined several times and found empty.

TABLE XIX.—Arthur P.

					c.c.							mm.*					
1	1902 Dec.	30	Ewald	65 m.	170	Clear	...	+ +	18	12	67	11	.	R.V.	0	0	
2	"	31	"	1 h.	76	"	...	+ +	15	10	68	11	.	"	0	0	
3	1903 Jan.	1	"	90 m	12	"	...	+ +	38	3	55	12	.	"	0	0	
4	"	1	Asylum supper	15 h.	0	"	...	...	...	...	.	.	..	..	.	..	Fasting stomach.

* Peptic value, Mett's method.

TABLE XX.—Wilhelmina D.

Number.	Date	Meal employed.	Time in stomach	Amount recovered.	Condition of stomach washings after removing test meal.	Acids. Lactic acid (Uffelmann).	Acids. Hydrochloric acid (Gunzberg).	Acids. Free hydrochloric acid.	Acids. Combined hydrochloric acid.	Acids. Total acid	Ferments, etc. Pepsin egg tablet digested in	Ferments, etc. Rennin coagulation of milk occurred in	Ferments, etc. Starch digestion (Lugol)	Ferments, etc. Bits of mucosa.	Ferments, etc. Important microorganisms	Remarks.
	1902		hours	c c.							hours	min				
1	Jan. 16	Ewald	1	130	Few small bits bread.	0	+	42	28	72	4	5	R	0	0	
2	" 17	"	1	115	Nothing	0	+	40	18	68	4½	5	R.V.	0	0	
3	Feb. 15	"	1	150	"	0	+	38	26	70	3	5	"	0	0	Ate 10 gms. more bread than before.

TABLE XXI.—Harrington W.

	1902		hours	c.c.							mm.*	min.				
1	Nov 30	Ewald	1	0	Clear	...	...	...	...	...	..	...	...	0	0	
2	Dec. 1	"	1	8	"	...	0	0	0	8	0	0	R.V	0	0	
3	" 1	Asylum dinner	3	3	"	0	0	0	...	..	0	0	...	0	0	
4	" 2	Ewald	1	5	"	0	0	0	0	0	..	...	R.V.	0	0	
5	" 2	Riegel	3	0	"	0	0	0	0	0	0	0	0	0	0	
6	" 1	Asylum supper	14	0	"	...	...	...	..	...	..		...	0	0	Fasting stomach.

TABLE XXII.—W. E. G.

	1902		hours	c c.							mm.*	min.				
1	Nov. 30	Ewald	1	34	Clear	0	0	0	0	2	0	0	R.V.	0	0	Excessive mucus.
2	Dec. 1	"	1	46	"	0	0	0	0	2	0	0	"	0	0	" "
3	" 1	Asylum dinner	3 5	68	"	0	0	0	0	10	0	0	"	0	0	" "
4	" 2	Ewald	1	82	"	0	0	0	0	15	0	0	"	0	0	" "
5	" 2	Riegel	1	10	"	0	0	0	0	10	0	0	A.C.	0	0	Mucus normal; stomach washings before
6	" 1	Asylum supper	11	0	"	.		..	.	.	...	...	..	...	..	Fasting stomach. [meal perfectly clear.

* Peptic value, Mett's method.

CASE III.—Harrington W. (see Table XXI.), aged sixty-three years, widower, American, farmer. No insanity in family. Admitted to asylum October, 1896. In the spring of 1896 patient complained of indefinite symptoms on the part of his stomach. He began to worry about financial affairs and about his health. He was hypochondriacal and thought himself unable to work. Delusions of health continued. In April, 1897, he thought his bowels did not move and that this was due to a growth occluding the passage. He begged for an operation. He refused to eat, wanted to starve himself. Forced feeding had to be instituted. He was markedly depressed. November 1, 1899, he attempted suicide. October 15, 1900, second suicidal attempt. Bowels regular.

Patient is well nourished.

Blood: Red cells, 4,000,000; haemoglobin, 70 per cent.; white cells, 5490.

Urine: Negative.

Weights: 1897, 170 pounds; January, 1902, 193 pounds; April, 187 pounds; June, 200 pounds; August, 196 pounds; November, 184 pounds.

CASE IV.—W. E. G. (see Table XXII.), aged forty-eight years, married, American, occupation unknown. Previous to his mental trouble patient was always of a cheerful disposition, quiet, and talked but little about his business.

In August, 1901, he began to cry; little other change was observed; several weeks after this he became much worried about the wood, and thought he and his family would freeze to death. Insomnia developed. His appetite was fair, but at times he believed something was wrong with his food. At one time he attempted to jump out of a window, as he thought the house was on fire.

Patient was admitted to the asylum March 10, 1902. He was very much depressed; he answered questions with great reluctance. He had hallucinations of hearing; would stop and listen to imaginary voices. He became very apprehensive, believed that other inmates composed an army brought to the institution to kill him. He was restless, constantly moving about the ward. His condition continued the same until May 29, 1902, when he was taken to his home for a visit. While there he secured a revolver and shot himself.

Physical examination entirely negative.

Stomach in normal position.

CONCLUSIONS. 1. In states of mental depression (melancholia) hyperacidity is the rule, occurring in 71.4 per cent. of von Noorden's 14 cases, 81.8 per cent. of our 22 cases, or in 77.7 per cent. of all cases (36 in number). Males and females are equally affected.

2. This hyperacidity is due to a true hyperchlorhydria. Hypertotal acidity also occurred in our cases, and in this respect they correspond exactly with those reported by von Noorden.

3. The hyperchlorhydria is of moderate degree, is fairly constant, and is associated with increased peptic power and rapid evacuation.

4. The increased secretion is due to the neurosis or psychosis and not to proliferative changes in the glands, as is evidenced by the presence of increased secretion associated with degenerative changes in the glandular elements, and of the entire mucosa.

5. The evacuation of the stomach is usually normal or somewhat hastened after the Ewald test breakfast, while after the large stimulus of the Riegel meal and ordinary Asylum meal it is more frequently hastened.

6. In the cases showing hyperchlorhydria the peptic value is never below normal and is frequently increased.

7. Many of the insane are suffering from various forms of gastro-intestinal disease. These conditions are very frequently overlooked, probably because complaints and delusions of the digestive tracts are so common in these patients. Our experience and the results of this investigation prompts us to urge the absolute necessity for systematic routine examinations of all the secretions and functions, of the body including what is often a very difficult task, the examination of the stomach contents. The signs of disease in the insane are almost wholly objective, and here more than in normal mental states it is essential that every modern method of value should be exhausted in order to arrive at a complete diagnosis of the case. Cancer and ulcer of the stomach may thus often be recognized in its incipiency. Chronic gastritis in its several forms, as well as the painful neuroses, will also often be encountered. If proper treatment be instituted it is not at all improbable that the mental symptoms in these cases will proportionately decrease.

SOME REMARKS ON THE NATURE AND CLASSIFICATION OF INSANITY.[1]

By Theodore Diller, M.D.,
of Pittsburg.

The ancient truism that a sound mind results from a sound body implies that the mind is not sound when the body is diseased. An unsound body may be expected to produce symptoms and physical signs by which its diseased condition may be recognized. Among the manifestations of the diseased body are those symptoms which are called mental. But mental symptoms are seldom or never the sole manifestations of bodily disease. Yet they are often so conspicuous as to dominate the clinical picture and lead us to lose sight

[1] A paper read before the Annual Meeting of the American Neurological Society held at St. Louis, September 17, 1904.

of the physical signs and symptoms which almost always, in a more or less marked degree, accompany the mental symptoms. The mental symptoms are commonly divided arbitrarily into two classes: those which constitute insanity and those which, because they are insufficient in number, degree, kind, or duration, are held to be inadequate to constitute insanity. In other words, it is commonly held that all departures from the mental norm do not constitute insanity, as, *e.g.*, the mental manifestations in fevers, Bright's disease, neurasthenia, chorea, etc. What and how many and how prolonged mental symptoms must be to constitute insanity are questions of individual opinion. We have had enough discussion of the subject of insanity to be able to reach the conclusion that it has never been and never can be defined in such a manner as to meet with anything like general acceptance.

Abnormal mental manifestations are measured by terms such as "severe," "prolonged," "insufficient," etc., as against "inches," "ounces," etc., in dealing with the purely physical; and it is therefore not to be wondered at that the term insanity is a variable and arbitrary one, since the standards of measurement are elastic and inexact; and description of mental symptoms must, therefore, always reflect the personal equation of him who makes it.

The term insanity is used to designate a more or less severe, prolonged, and comprehensive group of mental symptoms, and for the reasons stated there is no way of differentiating it sharply from the great mass of mental symptoms which in general are not held to constitute insanity. And yet many observers have felt that they *must* decide as to whether the mental symptoms present in a given individual constituted insanity or not, as though insanity were a clinical entity which could always be recognized if we are only keen enough to do so; and so it always can be, according to the observer's own standard of insanity, which is never exactly like that of any other observer.

Insanity, a large group of mental symptoms, is treated as though it were itself a grand division of disease, capable of division into groups and sub-groups. As the range of the normal mind is infinite, so is that of the abnormal, and the attempts to classify insanity have always been futile, no two authors ever agreeing quite as to what must be present or absent to constitute it, or as to the exact characteristics of any given group.

For the purely scientific study of mental symptoms, the varying needs of legal requirements should be wholly disregarded. Attempts to formulate definitions of insanity for the law have clouded the whole study of abnormal mental manifestations.

Happily there is a breaking down of some of the artificial and arbitrary distinctions by which varieties of mental diseases were formerly erected. For example, mania formerly regarded by many as a well-established disease is no more such than is dyspnœa, or

hallucinations, or œdema, or palpitation. It is a symptom-complex dependent, like dyspnœa, upon a great variety of causes, and it may be a marked, prolonged, or intermittent symptom, or the reverse. It may be the main symptom or one of many symptoms of disease. The same may be said of melancholia, dementia, paranoia, etc. I have not been able to convince myself that we have gained much of value by hitching the maniacal and melancholic symptoms together under the designation of maniac-depressive insanity. An unprejudiced statement would be that the patient presented maniacal, melancholic, or paranoic symptoms, just as we now say he has delusions or hallucinations or obsessions.

A great fundamental error which has given rise to many others arises from the attempt to divide disease into mental and physical, as though they were not only separate and distinct divisions of it, but even dependent upon different causes. It has caused us to lose somewhat the sense of unity of disease as a whole, and has created a distinction which is artificial and non-existent. All disease generally speaking, manifests itself both by mental and physical signs and symptoms. How common are the minor (to say nothing of the major) mental symptomatic expressions of physical disease. On the other hand, even in such a gross disease as a brain tumor, has been found to be the cause of insanity. Head, in a painstaking article,[1] has made a series of interesting observations on the mental symptoms noted in a variety of physical disease. Similar investigations by Gamble[2] led him to similar observations.

This study of mental symptoms in so-called physical disease is a comparatively untilled field of inquiry, from which we may hope to learn much by further investigations. When mental symptoms are observed in well-recognized physical disease, as, *e. g.*, brain tumor or arteriosclerosis or meningitis, we do not say that they constitute insanity, even though it would be called such *if the physical disease were unknown.* While in some diseased conditions physical symptoms are conspicuous and mental symptoms but slightly in evidence, in other cases the symptomatic expression is just the reverse; but in the latter class of cases insanity is never the sole expression of disease. Some physical signs, such as altered pulse or respiration or tongue or skin or muscular movements, always accompany them. The absurdity of considering a group of mental symptoms as a disease becomes more obvious when we consider how to many of them various authors have attached physical symptoms or signs.

[1] Brain, 1902.

[2] Johns Hopkins Hospital Bulletin, July-August, 1904. Among Gamble's observations were the following: Hallucinations of sight in a case of mitral stenosis; hallucinations of smell in a case of asthma; hallucinations of hearing with alternating depression and exaltation in a case of adherent pericardium; alternating depression and physical exaltation in a case of mitral stenosis with failing compensation; loss of attention and memory and fears in a case of asthma with emphysema; loss of attention and memory, and confusional ideas in several cases of arteriosclerosis.

Symptoms of disease of all kinds then are to be referred to altered or defective or malformed physical structures in which the organ of the mind—the cerebral cortex—must share when mental symptoms are present. That the exact physical basis for the explanation of many mental symptoms is unknown does not invalidate this statement. In such cases such a basis must be postulated.

Indeed, I believe we are making some advance toward this position. Paresis, formerly accounted an insanity, is now recognized as a physical disease, presenting striking physical as well as mental symptoms; and it is now, as should be the case, described in text-books on the practice of medicine. Krafft-Ebing, in his text-book on insanity, significantly describes in a separate section, "Brain Diseases with Predominating Psychic Symptoms," under which he includes besides paretic dementia, acute delirious mania, cerebral syphilis, and senile dementia.

The painstaking observer is more and more impressed with the number of physical signs and symptoms observed in so-called insanities. In so-called acute delirious mania and conditions approaching it, the dry tongue, the sordes of the teeth, the elevated temperature, and the altered urine, all speak for a form of auto-intoxication with marked metabolic changes. In not a few of the so-called physical diseases, as, for example, Bright's disease, arterio-sclerosis, multiple neuritis, syphilis, and cardiac disease, the mental symptoms may be among the most striking presented by the disease.

Since insanity is a term which is indefinable and one to which a stigma of disgrace is attached, and since its acceptance or recognition tends to make it something other than an expression of physical disease, malformation or defect, it would be well if, as suggested by Dr. Richard Dewey, the term could be abandoned as serving no useful scientific purpose and as tending to place the investigation of mental symptoms on a false basis, and if the term psychosis were used in its stead and to include all abnormal mental manifestations. But we can hardly hope to dismiss a term used for many ages. Something might be gained, however, if the term were written between quotation marks to denote its infinite and indefinable character.

But it may be objected if the term insanity, with its division into groups, be abolished or side-tracked, we shall still have the symptoms and physical signs which go to make it up. True enough; but we shall be in better position to study them from the anatomical, physiological, pathological, chemical, and psychological basis. Then we will be in better position to study each case of disease on its own merits, without the forced necessity of placing it in some artificial classification. The patient with maniacal symptoms will not be called a case of mania and dismissed with this label, for we know that this symptom may arise from Bright's disease, in acute intoxica-

tion, or exhaustion, as an episode in paretic dementia or paranoia, and in many other conditions. We will search more thoroughly for its underlying basis.

But like the term insanity itself, some of its forms have names which are in general use, like paranoia, dementia præcox, etc.; and it must at once be admitted that these cannot readily be dismissed, even if it were desirable to do so. Let them then be used as arbitrary, tentative, and temporal names for certain groups of symptoms, not as names of diseases. But let us by studies of individual cases avoid the danger of attaching undue importance to a mere name. Let the name not be a stumbling-block, a hindrance, a check to individual study of symptoms and signs as they exist in individual cases.

The whole of the study of mental symptoms as a partial expression of disease would be immeasurably advanced if the patients exhibiting them were treated in the same hospitals with patients who suffer from so-called physical disease. Thus the profession and the laity alike would have an objective lesson which would teach them the unity of disease, and do more than anything else to remove the stigma of reproach from the term insanity and lessen the employment of the term itself, and, furthermore, do much to place the study of mental symptoms themselves on a proper basis.

One or two general hospitals in every city should have a few wards properly equipped to deal with patients who exhibit marked mental symptoms. Patients of this kind, like those of any other class, should not be received or retained after they have become chronic. In a quiet, unobtrusive way, St. Francis Hospital, of Pittsburg, has for many years provided four such wards, and the writer can personally testify that they have served a highly useful purpose, and justify the statements just set forth. We know no other general hospital in this country which makes a similar provision. A few general hospitals abroad provide such wards for patients with mental symptoms, the splendid new hospital at Nuremburg being the latest so equipped.

It is in our opinion highly desirable that such provision should become very general, and recent expressions of opinion seem to indicate that the day is not far distant when this desideratum will be attained.

THE RELATION BETWEEN CLIMATE AND HEALTH, WITH SPECIAL REFERENCE TO AMERICAN OCCUPATION OF THE PHILIPPINE ISLANDS.[1]

By William S. Washburn, M.D.,
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The purpose of this paper is to discuss the relation between climatic conditions and health, with special reference to Americans in the Philippine Islands. The subject involves a study of climatological reports, medical literature, and statistics. Climatic laws, especially those relating to the tropics, will first be briefly reviewed.

The Director of the Philippine Weather Bureau, Rev. José Algué, clearly expresses the meaning of the term climate in the following definition: "The climate of a country is the complex of the average atmospheric conditions which have an influence upon organic life; therefore, an adequate study of the different elements which go to make up these atmospheric conditions would give a true idea of the climate. The more important elements which form the climate may be reduced to three—viz., heat or temperature; water vapor in its different forms, such as humidity, evaporation, clouds, and precipitation; and finally, the movement or circulation of the atmosphere, which includes the direction and force of the wind, cyclonic and non-cyclonic storms, together with local and general storms."

While temperature is the fundamental phenomenon of climate, several different elements form the basis of its classification, and each classification is affected or modified by other elements or influences. For example, on the basis of temperature we have hot, mild, temperate, and cold climates; on the basis of the influence of continents and oceans, climates are classified as oceanic, insular, and continental; on the basis of salubrity, as healthful and unhealthful.

On account of the spheroidal shape of the earth, temperature varies generally with latitude, but other elements, such as elevation, the presence or absence of vegetation or of large bodies of water, movements of the atmosphere, water vapor in the atmosphere, marine currents, and configuration of land, bring about sharp contrasts in the same latitude, and have a marked influence on animal or vegetable life, or both.

About five degrees each side of the equator is a zone of calms and almost daily thunder showers; beyond this to twenty degrees, a zone of periodical rains in summer, in which zone lie the Philippine

[1] A paper read at the Second Annual Meeting of the Philippine Islands Medical Association.

Islands, characterized by a wet and a dry season; from twenty to twenty-eight degrees, a subtropical zone, in which are the deserts of Sahara, Arabia, and of Northern India, and the arid region of Mexico and Lower California; from twenty-eight to forty-five degrees, a belt of semi-periodical rains, characterized by four seasons and prevailing sunny days (the United States lies principally in this belt).

With respect to elevation above the sea level, there is an average variation of about one degree of temperature for every 300 feet, which is well illustrated by the difference in temperature between Manila and Baguio, Benguet province.

Forests and other vegetations and large bodies of water tend to counteract the effects of terrestrial radiation, and thereby to equalize local climates. On the other hand, a region barren of trees and other vegetation permits wide daily fluctuations of temperature, the days being hot and the nights cold, which become extreme in barren places and arid regions, rising to a mean summer temperature of 95° in Arabia, Persia, and Punjab, India; in the Punjab 125° in the shade has been observed. In a country covered by forests and other vegetation, and in proximity with large bodies of water, the total heat of the day is quite equally distributed over the twenty-four hours, the heat of the day not being excessive, even in the tropics, as illustrated by climatic conditions in the Philippines.

Temperature is greatly modified by movements of the air or winds, which constitute an important climatic influence by increasing evaporation, thus rendering the soil drier, removing dampness, and affecting the "sensible temperature," that is, the apparent temperature which we feel or the physiological temperature. In the tropics, practically covered by the longitudinal extent of the Philippine Islands and from six to twenty degrees north latitude, is a zone of trade winds called monsoons in the Indian Ocean and adjacent portions of the Pacific. Cyclonic movements of the atmosphere, characterized by regularity of course, are called hurricanes in the West Indies and the Indian Ocean, cyclones in the Gulf of Bengal, typhoons in the China Sea, and baguios in the Philippine Islands. The central and northern portions of the Philippine Archipelago lie in the path of these baguios or cyclones, but these regions are free from the cold, raw winds frequently experienced in New England and Canada, in connection with the course of cyclones in that region. Due to similar cyclonic atmospheric movements, the entire Mediterranean region is subject to violent changes of temperature in winter. The central portions of the United States, considerably distant from the sea, are also subject to violent atmospheric changes, where a day that dawns with the ethereal mildness of spring may end in the freezing coldness of winter..

The water vapor of the atmosphere constitutes an important climatic element. The atmosphere contains a variable quantity of

moisture, resulting from vaporization of water through the action of heat rendering it lighter than air. The higher the temperature of the air the more water vapor it can carry, and when increased to the point of saturation, if the temperature is lowered, condensation occurs and results in precipitation in the form of rain or dew, clouds or fog. A rise of temperature, therefore, renders the air drier, and a lowering of temperature, more moist. The greatest amount of evaporation being in equatorial regions produces the equatorial and polar atmospheric currents, which have an important climatic influence on contiguous land bodies. The average quantity of annual rainfall reaches the maximum in the tropics, while the *number* of cloudy or rainy days gradually increases from the equatorial regions toward higher latitudes, and decreases from the coasts to the interior of continents.

The heated waters of the tropics, flowing away from the equatorial regions, are replaced by the colder waters of the polar currents. Moreover, the cold produced by radiation over the surface of the ocean and land adjoining lowers the temperature of the water, which sinks to greater depths, so that, even in the tropics, deep sea observations show the water below the surface to be increasingly cold toward the depths of the sea, where it approaches the freezing point. Local interisland currents, as the Straits of San Bernardino and Surigao, materially modify the temperature of neighboring regions. The following are examples of the influence of marine currents: Washington, D. C., with cold winters and hot summers, is in the same latitude as San Francisco, whose mild winters and comparatively cold summers are due to the influence of the Japan current modified by its sweep across the Northern Pacific. On one side of the Atlantic Ocean is Labrador, treeless and cheerless; on the other side, in about the same latitude, are the British Isles, with abundant vegetation and a comparatively mild climate, which is due to the influence of the northern branch of the Gulf stream. Labrador is subjected to the influence of the cold Labrador or polar current.

In the classification of climates, based on the size and extent of masses of land, oceanic, insular, and continental, the climate of the Philippine Islands is largely that of the first two classes, oceanic and insular. Nowhere is the land distant from the seacoast more than sixty miles. The moderating influences of the great bodies of sea water are therefore operative. On account of climatic influences, the climate of the Philippines is widely different from those of tropical Africa, South America, and Asia in the same latitude. As a rule, the smaller the island the more equable the climate throughout the day and the year. The climate of the greater part of the Philippine Archipelago is for this reason comfortable and hygienically favorable for the treatment of many diseases. In the temperate zone an insular, mild, or equable climate is frequently a health resort.

Observations of the Manila Observatory show that while the temperature as indicated by the thermometer at *sea level* is practically the same throughout the Philippine Archipelago it is higher in some regions than in others during the months of April, May, and June, which cover the period of greatest heat.

During this season there is a mean temperature of about 87° in the northern part of the island of Panay and throughout the Island of Luzon, except the highlands and a portion of the provinces of Pampanga, Bulacan, Rizal, and Bataan.

During this period there is a mean temperature of about 83° in the parts of Luzon excepted above, including Manila and the islands of Romblon and Cebu, the peninsula of Zamboanga, and the west coasts of Samar, Negros, Panay, and Bohol.

A comparatively mild mean temperature of about 80° prevails during this period over the Sulu Archipelago, the extreme southeast of Luzon, and the eastern portions of those islands south of Luzon and contiguous to the Pacific Ocean—viz., Samar, Leyte, and Mindanao. If the readings of the thermometer for the elevated regions were included, these averages would be very considerably reduced.

Except for the months of April, May, and June, in the first and second regions named above, the mean temperature for the remaining part of the year at or near the sea level is about 80°. The observations of the Manila Weather Bureau further show that the annual variation of temperature is greater in the northern than in the southern part of the archipelago.

Some portions of the Philippine Islands are characterized by a rainy season and a dry season, the rainy season gradually beginning in June, being excessive usually in July, August, and September, and gradually decreasing to November; while in other parts of the archipelago there is a more even distribution of rain. Parts of the eastern coast of Northern Luzon and the interior of Luzon above 4000 feet, the Pacific Ocean side of the archipelago, including the eastern portions of the islands of Masbate, Panay, and Bohol, are subject to the heaviest rainfall, amounting to over 100 inches annually. In the other parts of the archipelago, including Manila, the mean annual rainfall is less than 100 inches, decreasing to about 35 inches at Zamboanga.

The geological formation of the Philippine Islands is of volcanic origin, and in places of moderate elevation the earth covered by cultivatable soil is not unhealthful.

The observations of the Manila Weather Bureau show a considerable difference in climate between the coastal regions of the islands of the archipelago and the elevated regions of the islands of Luzon and Mindanao. Mountain climate in these two islands is similar to that of Baguio, Luzon, whose altitude is 4777 feet and whose average temperature and humidity are relatively low during the hot months of March, April, May, and June, owing to the prevailing winds blowing from the China Sea during that season. The

climate of this region is ideal from November to June. The region consists of a rolling table-land well watered. The forests are of pine, and the soil productive. The vegetation is similar to that of the temperate zones. The following table is made up from observations at Baguio covering a period of two or three years:

Month.	Temperature (Fahrenheit).			Humidity (per cent.)	Rainfall (inches).	Rainy days.
	Mean.	Maximum.	Minimum.			
January	63.14	75.38	49.28	78.1	0.92	4
February	64.08	73.94	15.68	77.3	0.21	1
March	65.66	78.44	51.68	76.3	1.09	3
April	68.01	80.24	54.86	75.5	1.66	6
May	67.46	78.84	52.52	83.9	9.42	18
June	66.38	77.72	58.10	89.4	15.92	26
July	65.84	73.76	56.30	89.4	15.83	28
August	65.12	75.74	54.68	92.2	42.89	31
September	65.66	74.48	57.20	90.5	17.81	26
October	65.66	75.92	55.40	85.7	6.65	11
November	65.84	75.56	52.16	79.0	2.75	10
December	64.94	75.02	54.68	81.2	3.19	10
Year	65.48	76.82	53.24	83.0	117.81	177

With a mean annual temperature of [illegible], an annual average humidity of about 79 per cent., and an average annual rainfall of about seventy-six inches, Manila occupies an intermediate position in the archipelago, climatically as well as geographically. The average annual rainfall is less than that of Baguio (118.04), Albay (110.90), Negros (102.02), Singapore (93.22), Bombay (92.72), and Hongkong (86.85), and more than that of Batavia (71.15), San Isidro (70.71), Iloilo (69.85), Calcutta (60.83), New Orleans (60.30), Porto Rico (59.45), Jolo (59.28), Tokyo (57.78), Havana (51.74), Cebu (47.18), New York (44.70), Shanghai (44.06), Washington, D. C. (42.90), Honolulu (39.22), Key West (38.20), Zamboanga (34.89), Chicago (34.00), and San Francisco (23.40).

In Manila the average amount of sunshine, measured by hours, and its distribution corresponds closely with the average clear and partly cloudy days for each month and for the year, as shown in the following table:

Month.	Average clear and partly cloudy days.
January	23.9
February	23.8
March	23.9
April	25.1
May	19.9
June	17.3
July	14.9
August	14.0
September	13.1
October	17.1
November	18.6
December	22.6
Total	234.2
Average	19.5

From this table it is seen that the minimum of clear and partly cloudy days in Manila occurs in September, and the maximum in April.

The climate of Manila is quite fully demonstrated in the following table, made up from observations of the Manila Weather Bureau covering a period of over twenty years, giving the mean monthly and annual temperature, the mean daily, monthly, and annual ranges of temperature, the normal average of the humidity, the normal amount of rainfall, and the average number of days of rainfall:

Month.	Temperature (Fahrenheit).					Mean humidity.[1]	Mean rainfall.[2]	Mean rainy days.
	Mean	Mean monthly max.	Mean monthly min.	Mean daily range.	Mean monthly range.			
January	77.18	89.78	64 22	13.68	25.56	77.6	1.14	5
February	78.08	91.22	61 40	15 30	26.82	74.2	0.39	3
March	80 42	93.74	65.30	15.48	28.44	71.8	0.73	3
April	83.12	95.90	69.44	16.02	26.46	70.7	1.12	4
May	83.84	96.98	72.86	12.60	24.12	76.7	4.00	9
June	82.40	95.00	72.68	10.80	22.32	81.4	9.75	16
July	80.96	92.30	72.32	9.00	19.98	84.8	15.00	21
August	80.96	91.40	72.14	8.64	19.26	84.8	14.22	21
September	80.78	91.94	72 32	8.28	19.62	85.5	14.75	20
October	80.60	91.94	70.52	10.08	21.42	82.7	7.55	16
November	79.16	89 96	68.54	10.26	21.42	82 0	5.37	12
December	77 36	89.78	64.94	11.16	24.84	80.7	2.29	8
Year	80.42	92.48	69.08	11.70	23.40	79.4	76.31	138

[1] From observations covering the period 1883–1902.
[2] " " " " " 1865–1902.

The normal monthly average relative humidity in Manila ranges from 70 to 85 per cent., with a normal average of less than 80 per cent., is lowest during the hot, dry season, when high temperature prevails, and highest during the rainy season, when more moderate temperature prevails. The quantity of water vapor in the atmosphere thus affects the diurnal and yearly ranges of temperature, and particularly the "sensible" temperature. Air saturated with moisture feels hotter than the thermometer actually indicates, and if near the body temperature the cooling process by evaporation is completely checked.

Besides the amount of humidity in the atmosphere, "sensible" temperature depends upon many other factors, important in the Philippine Islands, not entering into temperature as recorded by the thermometer—viz., movements of air, exposure to direct rays of the sun, reflected heat, one's physical and mental condition, clothing, occupation, food, etc., variations in which tend to produce comfort or discomfort. These factors are given detailed consideration in another part of this paper.

On the following page is given a comparative table between the United States weather districts and Manila, with respect to humidity and precipitation:

Districts.	Relative Humidity.						Precipitation.							
			Departures.						Departures.					
	Normal	1903	1903 from U. S. normal	1903 from Manila	1903 from Manila normal	U. S. normal from Manila 1901	U. S. normal from Manila normal	Normal	1903	1903 from U. S. normal	1903 from Manila	1903 from Manila normal	U. S. normal from Manila 1901	U. S. normal from Manila normal
New England	77	77	0	-3.3	-1.6	-2.5	-1.6	47.8	35.7	-12	-1.9	-76.9	-41.0	-72.6
Middle Atlantic	74	74	0	-6.4	-1.6	-5.5	-1.6	43.8	43.4	-0.4	-1.2	-31.4	-10.2	-31.0
South Atlantic	77	76	-1	-1.1	-2.6	-2.5	-1.5	52.0	47.6	-4.4	-2.0	-24.0	-22.0	-23.6
Florida Peninsula	79	78	-1	-3.2	-2.6	-0.5	-0.1	51.0	48.1	-2.9	-2.5	-23.9	-23.0	-21.6
East Gulf	75	76	1	-1.2	-2.6	-1.5	-1.6	56.0	47.0	-9.0	-4.4	-25.6	-28.0	-19.6
West Gulf	74	74	0	-1.6	-2.6	-1.5	-1.6	47.2	37.1	-10.1	-2.1	-25.1	-10.4	-22.1
Ohio Valley and Tennessee	72	73	1	-1.4	-2.6	-2.5	-1.6	45.1	37.6	-7.5	-3.2	-26.2	-24.3	-20.1
Lower Lakes	74	76	2	-1.2	-2.6	-2.5	-1.6	39.4	35.3	-4.1	-5.1	-40.3	-14.6	-40.2
Upper Lakes	77	76	-1	-1.2	-2.6	-2.5	-1.6	32.2	31.6	-0.6	-2.0	-41.0	-25.4	-41.1
North Dakota	72	72	0	-2.8	-5.6	-7.5	-5.6	21.2	21.1	-0.1	-19.7	-43.3	-45.1	-56.5
Upper Mississippi Valley	72	74	2	-0.4	-4.6	-7.5	-5.6	24.2	24.3	0.1	-6.4	-41.4	-19.7	-41.2
Missouri Valley	69	72	3	-2.4	-6.6	-10.5	-9.6	24.7	22.2	-2.5	-3.4	-47.1	-35.1	-46.9
Northern Slope	61	64	3	-6.4	-10.6	-14.5	-13.5	22.4	22.6	0.2	-14.0	-43.9	-31.8	-43.2
Middle Slope	62	66	4	-2.4	-12.6	-17.5	-16.6	22.2	22.0	-0.2	-14.6	-53.6	-31.8	-53.4
Southern Slope	62	64	2	-10.4	-14.6	-17.5	-16.6	37.2	33.4	-3.8	-17.2	-32.2	-20.8	-32.4
Southern Plateau	39	39	0	-23.4	-23.6	-19.5	-21.6	10.4	9.3	-1.1	-21.3	-63.3	-32.5	-63.1
Middle Plateau	36	33	-3	-21.4	-23.6	-19.5	-23.6	11.7	9.5	-2.2	-23.1	-67.1	-32.7	-64.3
Northern Plateau	62	63	1	-11.8	-13.6	-17.5	-16.6	15.6	12.7	-2.9	-27.9	-62.9	-38.1	-59.9
North Pacific	80	80	0	-3.2	-1.4	-0.5	-1.1	51.9	43.7	-8.2	-3.1	-31.9	-22.1	-33.7
Middle Pacific	71	69	-2	-5.4	-3.6	-3.5	-3.6	29.4	21.0	-8.4	-16.6	-51.6	-38.6	-46.2
South Pacific	68	68	0	-6.8	-10.6	-11.5	-10.6	11.5	10.3	-1.2	-20.3	-63.3	-43.5	-61.1

	Normal (1)	1903	Manila 1903 from normal	Manila 1901 from normal	Normal (2)	1903	Manila 1903 from normal	Manila 1901 from normal
Manila	78.6	71.8	-3.8	0.9	75.6	10.6	-25.0	8.4

[1] From observations covering the period 1883–1901.

[2] From observations covering the period 1865–1901.

The following is a comparative table with respect to temperature, humidity, and precipitation between Manila and five other tropical cities in the Far East:

City.	Latitude.	Temperature.			Relative humidity.	Rainfall.	
		1901. Max. mean.	1901. Min. mean.	Annual normal.	Annual normal.	Annual normal.	Rainy days.
Singapore	N. 1° 17′	86.7	74.3	79.90	80.0	93.22	174
Rangoon	N. 16 46	89.3	72.7	79.06	87.0	98.89	116
Calcutta	N. 22 23	86.3	70.7	77.85	82.0	60.83	85
Bombay	N. 18 58	85.5	74.7	79.88	78.0	82.72	76
Batavia	S. 6 8	82.9	73.0	78.80	82.8	71.15	156
Manila	N. 14 35	88.2	72.1	80.40	78.6	75.61	138

As early as 1797 Heberden, and later in 1863 Scoresby-Jackson, conducted investigations in England and Scotland, respectively, on the influence of cold on health, and reached the conclusion that "mean temperature and mortality from all causes had an inverse relationship below 50° F."

From the investigations of Buchan, Mitchell, and Pettenkofer, the following general propositions were deduced:

"1. A preternaturally dry air, with a high temperature, predisposes to the development of fevers and intestinal diseases.

"2. A very moist atmosphere, accompanied by a low temperature, is likely to induce bronchial and rheumatic affections.

"3. In summer and autumn the tendency to sickness and death is chiefly connected with the digestive organs.

"4. In summer and autumn a rise of mean temperature above the average increases the number of cases of and mortality from diseases of the digestive organs.

"5. A cool and rainy summer controls the prevalence and fatality of diarrhœal diseases."

The effects of temperature in producing disease are often confounded with the change of temperature, the latter of which is, perhaps, the more important of the two.

The literature with reference to the effects of tropical climates upon the human body appears to be even more exhaustive than that applicable to the temperate zone.

It has been shown that the presence or absence of vegetation affects temperature, and that the condition and character of soil have an important relation to health, the influence of which is exerted mainly through the media of water and air.

Twenty years ago Parkes defined the effects of climate upon the human body to be "the sum of the influences which are connected with the solar agencies, the soil, the air, or the water of a place," and stated that: "The deaths in the West Indies were partly owing to the virulence of yellow fever . . . and the general excess of

other febrile and dysenteric causes. The simple precautions which are efficacious in England have been as useful in the West Indies. Proper food, good water, pure air have been supplied, and, in proportion as they have been so, the deadly effects attributed to climate have disappeared. The effect of a tropical climate is, so to speak, relative. . . . Take away these sanitary defects, and avoid malarious soils or drain them, and let the mode of living be a proper one, and the European soldier does not die faster in the tropics than at home."

Billings, writing at that time, and Williams at a later period, expressed substantially the same views.

Some authorities take the view that in certain portions of the tropics and under favorable conditions, acclimation is possible; this group includes Quatrefages, Bertillon, Treille, van der Berg, Overbeck de Meyjer, Schellong, Daübler, and Koch. Daübler says that "Health in the tropics depends upon tropical hygiene and ethnic adaptability or acclimation, the manner of living playing the most important role." Stockvis and Below say without qualification that adaptation of Europeans to conditions in the tropics is possible.

It is admitted that colonization has been successful in Queensland, Australia, lying between the parallels of fifteen and twenty-eight degrees, south latitude, a region partly tropical and partly subtropical, which increased in white population from 28,000 in 1856 to 343,000 in 1886. Queensland has as low a rate of mortality as Europe.

Daübler concluded that European colonization has been successful in certain subtropical and bordering tropical regions. Virchow, Carey, Cosales, and Stanley give statistics in which they state that the European may, under proper sanitary conditions, transplant himself anywhere. Thomas says: "Under the tropical skies the climate is not an obstacle. The soil remains the only enemy we have to conquer." As to whether vital energy is sustained in the tropics, Stockvis says: "Not only is European acclimation in the tropics possible, but also full colonization," his statement being based on "results of exact observations in the laboratory at Batavia, as well as upon statistics of European colonization in South America." Felkin was able to conclude from his observations in Africa that "The adaptation of the white race to a tropical climate is possible," and Calvert concludes that it is practicable.

Nevertheless, the proposition early laid down that it is impossible for the white race to adapt itself to a tropical climate is supported by Lind, Saulnier, Divivier, Boudin, Helfft, Hirsch, and Rower. Munson expresses similar views, but states that "It is not at all easy to say how much of the excess of mortality of Europeans in tropical and subtropical countries over that of the natives is simply attributable to climate *per se*, and is consequently inevitable, or how much of it is the direct consequence of unsuitable habits of life and of the

neglect of sanitary laws and of personal hygiene." Munson's statement to the effect that there is an excess of mortality among Europeans in tropical and subtropical countries over that of the natives does not appear to be borne out with respect to the Philippine Islands by the last published report of the Board of Health, which shows the death rate in Manila for the twelve months prior to September 1, 1903, as follows: Filipinos, 43.42 per cent; Chinese, 28.26 per cent.; foreigners, 18.72 per cent.; Americans, 14.80 per cent. Excluding cholera, the death rate for Americans for the same period was only 3.83 per cent. The comparative death rate of natives in the army seems to corroborate the report of the Board of Health in this matter. While it is true that the native is less susceptible to a few diseases than is the white race, the evidence is cumulative that the rate of mortality among the white race now living in the tropics is less than that of the native population.

Conclusions as to the unhealthfulness of tropical climates appear to have been drawn principally from statistics relating to military organizations, and proper consideration does not appear to have been given to the fact that in many parts of the tropics colonies of representatives of Northern as well as of Southern Europe have lived for years or for generations in comparative health and comfort in India, in the straits settlements, in the East Indies, and in the West Indies. This statement is amply verified in the history of cities of Southern Asia, including Bombay and Calcutta, and of the islands of the East Indies, including Manila.

Referring to the problem of the relations of climate to health, Calvert sagely observes that "Statistics of military operations possess practically no value in an inquiry of this nature, for troops campaigning in any country are most liable to diseases."

The classical proposition that a white man cannot adapt himself to a tropical climate appears to be based partly on the experience gained in former times through exploration and attempts to colonize in the tropics. The results were generally disastrous and have not been forgotten, being constantly referred to as conclusive evidence of the impossibility of colonization by the white race in the torrid zone. Disease and death invariably accompany the invasion of an army into any country, whether it be in the temperate or in the torrid zone. It is likewise true that, while great heat is borne by the system if the body temperature is kept down by abundant perspiration, and if the hot season is not of long duration, protracted residence in a region of *great* or *intense* heat appears to exercise a depressing influence, "lessening the nervous activity and impairing the great functions of digestion and respiration and sanguification and probably the power of forming new and healthy tissue." There are many parts of the tropics, however, where the heat is not intense at any period of the year, or where the season of intense heat is not of long duration, in which the evidence is cumulative that the white

race can live for an extended period without forfeiture of health or longevity. In the tropics there has been a steady decrease in the amount of illness and in the death rate among Europeans in the colonial possessions of the British, Dutch, and French. There has been no material change in the climate either of the temperate zone or of the tropics. As the climate has not materially changed, the reduction many fold in the amount of illness and the mortality rate must be accounted for largely by the observance in the tropics of the hygienic measures of modern times which obtain in the temperate zone.

There has been improvement in health conditions in the United States, just as there has been more recently in the tropics, with improvement in hygienic living and surroundings. A portion of the tropical world will probably continue to be unhealthful for Europeans until there can be observance of the rules of personal, domestic, and public hygiene. Insular and other portions of the tropics, where the temperature, relative humidity, and soil conditions are favorable are not shown to be essentially unhealthful.

For the period prior to 1900 there are "no statistical records of sanitary value" available for comparison of health conditions in the city of Manila or the Philippines. The report of the Commissioner of Public Health for the year ended August 31, 1903, shows a steady improvement in general health conditions. Excluding cholera, which the Commissioner states "is to be regarded as an exotic in the Philippine Islands," the *general* mortality rate decreased from 39.66 per cent. in 1900 to 29.45 per cent. in 1903. The death rate among Americans of 3.83 per cent. for the same period and of 1.82 per cent. for the year ended August 31, 1904, were very much lower than the *general* mortality rates in Manila or most cities in the temperate zones. The American population, however, does not include the usual proportion of old and young persons, and some have returned to the United States on account of illness. From the best information obtainable, the mortality rates of infants and of old people among the whites are not greater than those in the United States, and the death rate of those who returned to the States is very low.

The mortality of infants under one year of age among the natives is shown to have been 40 per cent. of the total number of deaths and to have exceeded "the combined mortality of Asiatic cholera, bubonic plague, smallpox, dysentery, malarial fever, typhoid fever, and beriberi." Attention is called to the fact that the *general* death rate in Ermita district, where reside a larger proportion of whites than in other districts of Manila, was 19.22 per cent., "about the same as that of the best modern cities." The excessive infant mortality among *natives* is due to the lack of proper care and food. The health reports show that of the total number of deaths in the city of Manila, approximately one-half occur "in the absence of any treat-

ment of a qualified physician." The high death rate among the natives appears to be largely independent of climatic influences, and accounts for the islands being so sparsely inhabited. The population of Java (area 51,961 square miles), with similar climatic conditions, increased from about 17,291,000 in 1872 to 24,133,685 in 1891, while the population of the Philippines, with an area of 127,863 square miles, more than double the area of Java, is approximately 7,000,000, an increase since 1887 of only about 1,000,000.

With respect to cholera, smallpox, malaria, and beriberi, the Commissioner of Public Health states:

"The Filipinos have been not only more liable to contract cholera than the Chinese or Americans, but, when they do get the disease, they are almost twice as liable to die therefrom. The number of Americans who contracted the disease is unduly high; but the cases occurred largely among soldiers who entered native houses and partook of native foods and drinks in defiance of standing military orders.

"During the twelve months prior to September 1, 1903, there were 99 cases of smallpox in Manila, with 16 deaths, or no more than occur from this cause in various cities of the United States of equal size having a large dark-skinned population.

"Malarial disease is by no means as common or dangerous in the Philippines as in many other parts of the tropics.

"Whites are relatively very insusceptible to the disease (beriberi) and but few cases occur among them."

Notwithstanding the unfavorable sanitary and hygienic conditions, statistics of the Board of Health show, by comparison of death rates for other cities, that Manila in 1903 was more healthful than were in the preceding year or two Bombay and Calcutta, and compared favorably with Hongkong, Moscow, Trieste, Naples, Madrid, Cairo, Alexandria, New Orleans, Charleston, Memphis, and other cities in the temperate zone.

Referring to the health of the army, the Surgeon-General states: "The sanitary improvements in the conditions of the soldiers have gradually lessened the rates of sickness and mortality since the war, and, although a large proportion of the troops have been on practically war service in the Philippine Islands since 1898, the rates of sickness and mortality for the calendar year 1903 more nearly approach those of 1897, when the low death rate of 5.11 per thousand of strength for the whole army was recorded, than at any time since the outbreak of the Spanish-American war. This is a most gratifying state of affairs, and it is hoped that with the present sanitary knowledge put to practical energetic use a further reduction in the rates of sickness and mortality in the army will be effected."

Commenting on discharges for disability, the report of the Surgeon-General states: "It is true that with the most rigid examination some physically unfit men will be accepted. It is believed, however,

that one-third of the recruits who were afterward discharged for disability during the year 1903 should have been passed physically."

The statistics of the Surgeon-General's reports do not give complete and exact data as to the comparative health conditions between American soldiers in the United States, American soldiers in the Philippines, and native soldiers in the Philippines. From information contained in the reports, however, it is inferred that while the amount of sickness in the islands among American soldiers and native soldiers is about the same, the mortality rate of the natives is higher than that of the Americans, and that at no equal period of time has the mortality rate on account of sickness in the Philippines been as high as it was during the Civil War or during the Spanish-American war.

Asiatic cholera, yellow fever, dysentery, malarial fevers, and their sequelæ, which have occurred extensively in the United States and elsewhere in the temperate zone, as well as in the tropics, are not the destroyers of former times. Yellow fever is not a factor in the health problem of the Philippines, and Asiatic cholera is now nonexistent in the islands.

As to malaria in tropical regions, the worst forms exist in Central America and along the banks of rivers and in great river deltas of Africa and South America. It is common in India, Southern China, and in most parts of the East Indies, rare in Japan, Australia, New Caledonia, some of the South Sea Islands, and practically unknown in the Hawaiian Islands, Samoa, and New Zealand. In Manila and the greater portion of the Philippines, where Americans are required to live, malaria does not seriously affect the health of the white man who observes the reasonable hygienic measures which are required to be free from this disease during part or all of the year in many portions of the United States. The difference between the care of troops in modern times and formerly is illustrated by an instance in Ceylon, where in former times it is shown that at a certain station out of a total strength of 254 men all contracted malarial fever and 205 died; whereas the admissions for this disease in Ceylon in 1898 were only 71 per thousand.

Dysentery and other intestinal diseases in the Philippines are becoming less frequent in proportion as there is increased observance of domestic hygiene. The important diseases which are limited to the tropics are few. The great proportion of the decimating diseases have been more or less common in both the tropics and the temperate zones. As increased temperature and humidity, however, favor putrefactive changes and the growth and development of micro-organisms, some diseases are more prevalent in the tropics than elsewhere. On the other hand, sunshine, with little moisture and periodic heavy rainfall, are health-promoting agencies. In the

greater part of the Philippines, as in the greater part of the United States, the question of climate in its relation to health appears to resolve itself principally into the ability of man to circumvent disease through sanitary and hygienic measures.

It has been demonstrated in the Philippines that bubonic plague can be controlled by preventive measures, although it is an extremely fatal disease, as is cholera. Fortunately, however, comparatively few Europeans in the Philippines were attacked by cholera during its recent visitation, and fewer still by bubonic plague, only those suffering from disease whose manner of living is most unsanitary or who fail to observe reasonable hygienic precautions.

Of three great ravaging diseases in the United States—viz., tuberculosis, enteric fever, and penumonia—tuberculous cases among Americans in the Philippines are less frequent than in the United States, and enteric fever and pneumonia are comparatively rare. In Manila for the year ended August 31, 1904, but two deaths occurred from enteric fever and two from pneumonia among the European or foreign population.

The greatly increased amount of sickness and death from intestinal diseases during the hot season in the United States, and from respiratory diseases on account of the cold, damp, and changeable weather of the winter and spring seasons, together with many diseases which are rare or unknown in the Philippines, but prevalent in the United States, are important considerations when comparing the healthfulness of the climate of the Philippines with that of the United States.

Among the more or less prevalent diseases in the United States which appear to be comparatively rare or unknown in the Philippine Islands are gout, rheumatism, rheumatoid arthritis, neuralgia, cancer, enteric fever, relapsing fever, rubeola, scarlatina, diphtheria, rabies, and nearly all diseases of the respiratory system.

The character of dwellings with respect to structure, arrangement, and sanitary features is important from the standpoint of health and comfort. A house cannot be said to be in a hygienic condition which is so constructed as to prevent the sun's rays from reaching every portion of the interior when desired. An old Italian proverb has it, "Where the sun does not enter, the physician will."

In a tropical climate a house properly constructed and located, with a sufficient space around it to ensure free circulation of air, will have at any time of day or night some portion comparatively cool and comfortable. As the relative humidity of the air is greater near the surface of the earth during the night, when the temperature is at or near the daily minimum, approaching the dew point, dwellings in the tropics should be so constructed that the sleeping rooms are as far above the ground as practicable. A considerable proportion, approximately one-third, of the volume of soil consists of air. It is

a common occurrence in the Philippines to observe a house built on damp soil with the subsoil water near the surface, a depository of organic matter, the inhabitants being subjected to the emanations from the soil, especially at night, through open floors, putrefaction being active in the soil.

While the so-called "miasma" and "effluvia" possess little value as definite etiological factors of disease, the high specific gravity of certain noxious gases renders their presence in the air, as well as that of organic matter, harmful to those who sleep upon or near the ground.

In the tropics especially, in addition to a proper sewage system, a sanitary abode seems to require good drainage, elevation, with the ground under and immediately around the house surfaced with concrete or other impervious material, tight floors, and thorough ventilation. The prevailing absence in the Philippines of these sanitary conditions is an important etiological factor of disease, closely allied to medical climatology, and is entitled to careful consideration in any discussion of climate in its relation to health.

As the surroundings of a dwelling should lend much to health and comfort, the ground should slope away from the house, carrying all surface water to a distance, and the intense light of the dry season be softened by the presence of lawns and shrubbery around the dwelling. A pleasing landscape with the soothing influence of its predominating color of green appears to have been regarded as of very little importance in Manila. It is unfortunate that in these islands, where the benefits of plant life are so easily obtainable with a minimum of effort, there are not more habitations surrounded by lawns and flower gardens.

Medical climatology also involves the matter of a pure and abundant water supply, which is an important factor in the healthfulness of any locality. Because of the pathogenic organisms which water frequently contains, many refrain from drinking a sufficient amount of wholesome water and incline to the excessive use of alcoholics. Hydrotherapy in the tropics seems to be particularly appropriate. It appears to be the concensus of opinion among medical men that, as a rule, the *quantity* of pure, wholesome water is of more value than the medicinal virtues of the water of this or that mineral spring. The frequent excessive use of alcoholics and the tendency to abstain from drinking water on account of the danger of contracting disease thereby has claimed the attention of investigators.

Wulffert, of Berlin, describes the effect of alcohol in the tropics upon the digestive organs, nervous system, and the heart, and comes to the conclusion that these organs are hampered by its use in their effort at adaptation to the hot climate, regulation of bodily heat being disturbed, excessive perspiration induced, and the organs weakened as to power of overcoming disease. Similar conclusions

are reached by other physicians, among them being Fiebig, Rudel, Anschutz, Schwalbe, and Breitenstein. Fiebig states that of 40 cases of "black-water fever" which he treated in the tropics, all were alcoholics. From his investigations, Wulffert concludes that the obstacles which oppose acclimation are those which are presented by local conditions; example, malaria, and those which lie in the colonizer himself; example, the alcohol habit, and that "a white people addicted to the alcohol habit can never hope to settle tropical countries," and recommends as preparatory measures for life in the tropics:

"1. Vigorous bodily work or sport in the open.

"2. The use of the system of diet recommended for the tropics by Rubner, a system which he considers as not unsuitable also to the temperate zone.

"3. Complete abstinence from intoxicating liquors."

Referring to the prevalence of alcoholism, Munson states: "In the military service the abuse of alcoholic liquors is one of the most potent factors by which efficiency is impaired, and in the past its effects were so apparent that drunkenness was largely looked upon as a distinctively military failing. Its influence as well as its prevalence is indifferently shown by military statistics; for all drinking men do not enter hospitals for alcoholism, and a certain proportion of drunkards succumb to such various diseases as gastritis, cirrhosis of the liver, and nephritis, in the development and ultimate result of which the use of intoxicants has probably played no minor part; further, the term alcoholism includes many pathological conditions and their symptoms, these varying according to the quantity of alcohol taken and the manner in which it is consumed."

In a climate like that of the Philippines there is little excuse for being deprived of an abundance of air in-doors, or for overcrowding and little ventilation. The conditions are favorable to living practically in the open air night and day.

Even in the time of Hippocrates the stated laws of health recognized a proper relation between food and drink and exercise, expressed by the formula, "temperance and exercise."

The climate of the Philippines is favorable, in that exercise can be taken practically in the open air at all times of the year, where properly constructed and healthful habitations are available. Abnormal physiological function in the tropics is believed to be due largely to the lack of physical exercise.

Parkes estimates that "every healthy man ought to take daily an amount of exercise equivalent to 150 tons lifted one foot, or a walk of about nine miles," which is practically equivalent to the average demands of a majority of trades and manual occupations.

In addition to shelter and surroundings, the influence of climate is further modified by the appropriate use of food and clothing.

Climatic conditions appear to be more favorable in the Philippines than in the temperate zone for the observance of the hygiene of clothing.

Notwithstanding the climate of the Philippines is commonly referred to as enervating, Americans connected with the public service, whether engaged upon work in offices or upon work requiring their presence in the open air, have probably on the average accomplished more in the islands than is ordinarily accomplished at home in similar lines of work. Regular habits, the leading of a temperate life, and the absence of indulgence in excesses, have much to do with one's health in any country. The climate of these islands should not be held responsible for illness or physical breakdown due to those excesses which tend to physical demoralization in the temperate zone. Absence from home environment and restraints probably accounts for a large number of Americans who, by plunging into excesses of various kinds, caused their own physical demoralization and failure.

It should be remembered that there is no place, even in the temperate zone, suitable for and agreeable to all persons. There are so great a variety and so many degrees of physical obliquity that no single combination of climatic influence, however favorable, can be found to meet all conditions. Because the climate of the Philippine Islands does not agree with some, even who take reasonable care of themselves, does not necessarily indicate that the climate is unhealthful. The physical condition of many improves after coming to the islands, and that of others remains normal.

As a general rule, the appearance of Europeans living in the tropics seems to indicate an anæmic condition. The "cry of the system for better blood," expressed in the form of neuralgia, is, however, rare. In this connection Cabot states: "'The most striking example of the fallacy of judging of anæmia by the color of the skin and mucous membranes is the so-called 'tropical anæmia.' Practically all persons belonging to white races who take up their abode in the tropics acquire, after a term of years, an extreme pallor of the skin and mucous membranes, and this appearance has usually received the title of 'tropical anæmia.' It turns out, however, from the careful studies of several different investigators, that the blood of such persons shows absolutely no anæmia or other variation from the normal. The appearance of the skin is probably due to the action on the peripheral nerves and vessels. Tropical anæmia is a condition not of the blood, but of the skin and subcutaneous tissues." These views are confirmed by the more recent studies of Phlen, Marestang, Gringus, and Eijkmann. Gottstein concludes that "as a result of long residence in the tropics neither a quantitative change of the blood nor a reduction in the number of its cells or its hæmoglobin-content takes place." These conclusions are verified

by the following results of examinations recently made by Mr. M. T. Clegg, under the direction of the Acting Superintendent of Government Laboratories, of twenty-three employés of the Philippine Civil Service, selected at random, who have been continuously in the Philippine Islands for over four years:

Age.	General health	Continuous residence (years).	Weight on arrival.	Weight at present.	No. of red blood corpuscles per cent. c. m.	Specific gravity of blood.	Hæmoglobin estimate per ct.	Color index
27	Feeling of debility during past three months	4¾	128	152	5,000,000	1060	100	1
32	Has feeling of debility at times	6	140	150	5,000,000	1060	100	1
33	Good	5½	155	150	4,800,000	1060	100	1
28	Has had typhoid fever and dysentery	6½	160	210	5,000,000	1060	100	1
33	Has had dengue	6	157	197	5,000,000	1060	100	1
31	Has had dysentery	6	158	123	4,000,000	1056	80	1
30	Has had typhoid	5½	137	135	5,000,000	1060	100	1
30	Malarial fever (1900)	5½	170	175	4,500,000	1056	80	0 9
30	Good	5½	160	172	4,800,000	1059	95	1
32	Good	6	180	160	5,000,000	1060	100	1
25	Good	6	160	150	4,500,000	1059	95	1
28	Fair	6½	132	134	4,500,000	1056	80	0 9
34	Good	5½	148	148	4,800,000	1059	95	1
30	Good	5¼	156	148	4,500,000	1059	95	1
38	Good	6	126	142	4,800,000	1059	95	1
33	Good	6	178	164	5,000,000	1060	100	1
33	Good	4⅓	170	182	5,000,000	1060	100	1
28	Good	6	140	135	4,500,000	1059	95	1
33	Good	6	145	154	4,500,000	1057	85	0 95
31	Good	6½	155	155	4,800,000	1059	95	1
	Eight days with cholera	4⅙	157	186	5,000,000	1060	100	1
	Good	6¼	120	145	5,000,000	1060	100	1
28	Good	6 11/12	185	156	4,800,000	1059	95	1
30 9	Average	5 8	152 9	157.5	4,786,956	1059	95	

NOTE.—Hammerschlag's method was used in estimating specific gravity and hæmoglobin.

Cantlie observes that about two years are required for acclimation of the white man in the tropics, after which good health is a reasonable expectation. Calvert makes a similar observation. These views seem to be borne out by statistics of the Board of Health of the Philippines,[1] which show the general average of sick days per year for five years for each American employé connected with the Civil Service of the Philippine Government as follows: First year, 4.80; second year, 7.63; third year, 4.47; fourth year, 0.81; fifth year, 0.03. Further corroboration is found in the following reported by Marston, showing the death rate of British white troops in India per thousand, based upon statistics for a period of eight years prior to and including 1884: First and second years of service, 6; third to sixth years of service, inclusive, 1.88; seventh to tenth years of service, inclusive, 0.73.

[1] Results of a Preliminary Study of the Health of Americans in Manila

The period of foreign service of the United States Army being two or three years, appears to cover practically the usual period of acclimation. The conclusion appears to be reasonable that the fourth and fifth years of residence in the Philippines are more agreeable from the standpoint of health than any of the preceding years, as is indicated by the Board of Health statistics, a large proportion of the personnel of the Philippine Civil Service having been recruited from officers and enlisted men of the volunteer establishment of the army. The views expressed by the president of this Medical Association, Dr. McDill, in his annual address, are in accord with conclusions reached by other observers.

The influence of the climate of the Philippines from a surgical point of view is clearly expressed by Major J. M. Banister, of the Medical Corps of the Army, in the following propositions:

"1. Aseptic results will just as surely follow aseptic methods in the Philippines as in the United States or Europe.

"2. Should septic infection occur in any clean case subjected to operation in the Philippines, blame the technique, not the climate.

"3. Successful attainment of the object for which the operation has been undertaken will follow careful and skilful surgery in the Philippines with the same regularity that we have been accustomed to observe in such work at home.

"4. Convalescence after surgical operations in the Philippines is rapid and satisfactory when such operations have been carefully and skilfully performed.

"5. The danger of mortality after a skilfully performed surgical operation in which all the details of a rigid aseptic technique have been carried out is not increased by the influences of the climate of the Philippines unless the patient is at the same time the victim of some other serious disease."

Similar views are expressed by the Attending Physician and Surgeon, Civil Hospital, in his report submitted in 1903 to the Secretary of the Interior.

Major-General Wood, Governor of the Moro Province, in his annual report to the Governor-General, states: "There is nothing in the climate of this portion of the island which prohibits a long residence here. The British residents of North Borneo and the Malay Peninsula and the Dutch in Java manage to live apparently very contented and healthy lives in those countries, which are hotter than any portion of the Philippine Islands. While the service is in a way severe, it does not seem to deter the average Englishman or Dutchman from competing eagerly for positions in the government of the colonies, and it is believed that Americans can live and do good work where any other white race can. A moral life, with plenty of hard work, will be found to counteract in most cases the so-called demoralizing effects of the Philippine climate."

The revelations of bacteriological research of the last few years, arming men with a knowledge of the causative factors of many tropical diseases, have diminished the importance of their geographical and meteorological relations, have destroyed theories of the climatic origin of disease, and thereby robbed the tropical climate of many of its terrors, such as yellow fever, malaria, Asiatic cholera, and dysentery. Among Americans the late lamented Major Walter Reed and his coadjutors did monumental work along this line in Cuba and the Army Laboratory at Washington. The preliminary work of the bacteriologists and pathologists connected with this magnificent and splendidly equipped new laboratory[1] in the Far East gives every indication that the research work already inaugurated will show brilliant results and add new names to the long list of investigators whose work and discoveries in the field of preventive medicine deserve the gratitude of the whole world.

CONCLUSIONS. 1. With respect to the principal climatological factors, temperature, humidity, and atmospheric movements, the climate of the Philippine Archipelago is not extreme, as it is distinctly insular in character, the greater portion of the land area being not far distant from the ocean. There are other modifying influences which obtain in portions of the archipelago, such as general oceanic and local interisland currents, prevailing winds, elevation, the state of cultivation and drainage of the soil, and the presence of forests and other plant life.

2. Excluding localities in the tropics characterized by excessive heat, high relative humidity, or unhealthful soil conditions, acclimation or physiological adaptation of the white man to tropical environment is possible.

3. If acclimation is possible, colonization is possible.

4. Failures of the white race to live in the tropics and maintain health, excluding localities indicated in the second conclusion, appear to have been due principally to non-observance of the rules of personal, domestic, and public hygiene.

5. As a rule, Americans appear to become acclimated in the Philippines during the third year of residence.

6. With sanitary surroundings and by observing the rules of personal and domestic hygiene, residence of Americans in the Philippine Islands appears to be attended with as little danger of disease and death as residence in the United States under similar sanitary conditions.

[1] The second annual meeting of the Philippine Islands Medical Association was held in the new Laboratory Building of the Bureau of Government Laboratories in Manila

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REVIEWS.

THE MODERN MASTOID OPERATION. By FREDERICK WHITING, A.M., M.D. Philadelphia: P. Blakiston's Son & Co., 1905.

THIS work is by far the most important publication which has appeared in the English language upon the mastoid operation. Its distinguished author has had such a vast experience that anything which he may write must command the respectful attention of those doing otological work. The book begins with a very interesting medical *résumé*, in which it is pointed out that the history of the mastoid operation is divided into two chronological periods, the first stage beginning when Rolfinck, in 1656, propounded the suggestion that the mastoid process might be artificially perforated, and the second period of the modern mastoid operation beginning in 1864, when Turnbull and Crosby in America and Ludwig Mayer in Germany each perforated the mastoid cortex for the relief of permanent mastoid process. The first suggestion for the operation was that the opening might serve as a means of relief in cases of deafness with excessive tinnitus, the idea being that an artificial vent thus formed might provide an "avenue for the escape of tumultuous noises." The author then proceeds to the history of the Schwartze operation, and concludes his historical *résumé* with a sketch of the development of the modern complete mastoid operation.

The second and by far the largest part of the book is comprised under the heading "Technical Portion." In it the author goes into the most minute detail as to the method of performing the operation, and the subsequent treatment of the operation wound. What he terms the complete mastoid operation differs from the Schwartze operation in two important particulars. First, that in order to provide for the complete removal of the mastoid apophysis it is necessary to perform a flap operation in making the incision through the skin, and, second, in the complete mastoid operation the pneumatic process and the diploic cells at the posterior root of the zygoma are entirely removed. The latter he considers a most essential measure. The description of the operation comprises many pages, and goes into every point which can be imagined in connection with it. The author's style is dogmatic and only justified by his very great experience and by the manner in which he adduces facts to support his contentions. The most striking features of the book are the illustra-

tions with which it abounds. These are excellently made from a cadaver. The one difficulty about them is that the dissections have been so well and carefully done that the conditions represented are too ideal to be looked for during the performance of an operation upon the living subject. The book is to be regarded as a most distinctly valuable acquisition to American otological literature, and its author is to be congratulated upon it. F. R. P.

LEITFADEN DER ELEKTRODIAGNOSTIK UND ELEKTROTHERAPIE FUR PRAKTIKER UND STUDIERENDE. VON DR. TOBY COHEN, Nervenarzt in Berlin. Mit 6 Tafeln und 39 Abbildungen im Text. Zweite vermehrte und verbesserte Auflage Berlin 1902. Verlag von S. Karger.

THE increased interest in the value of electricity as a diagnostic and therapeutic agent, which has developed in recent years among the medical profession, will make this manual of especial interest. It consists of 158 pages in the text, and seems to be written with the special object of leading the beginner by graded steps from the known to the unknown, yet it is sufficiently thorough to make it of value to the more advanced worker, as a handy reference book. It is mostly a practical work, though the theories for each effect are briefly given.

The plates consist of diagrams showing the muscles of sections of the body, with the most favorable points for application of the electrodes. The general rules governing the therapeutic applications are first given, and this is followed by a description of the special applications. As a whole, it well fills the demands of the practical work of the student and practitioner. G. E. P.

THE AMERICAN YEAR BOOK OF MEDICINE AND SURGERY FOR 1905. A Yearly Digest of Scientific Progress and Authoritative Opinion in all Branches of Medicine and Surgery, etc. Under the editorial charge of GEORGE M. GOULD, A.M., M.D. In two volumes. Volume I., including General Medicine; Volume II., General Surgery. Philadelphia and London: W. B. Saunders & Co., 1905.

As usual this Year Book fully fulfils the promise of its title-page, being a complete digest of the world's best medical literature, with critical comments written by thoroughly competent men in all branches of medicine and surgery. It is impossible to dig out for individual criticism any particular portion of the work; it is only

necessary to say that it should be on the shelves of every man who wishes to keep abreast with the progress of the profession.

F. R. P.

Traite 'd'Hygiene Procedes Rapides de Recherche des Falsifications et Alterations. Par le Dr. P. Smolensky (De Saint-Petersbourg). Fraduction du russe par S. Broido et A. Zaguelmann. Annotée par L. Guiraud et A. Gautie. Avec 119 Figures, pt. 752. Paris: G. Steinheil, editeur, 1904.

Of the 681 pages of actual text which this book contains, 531, or nearly four-fifths, are devoted to foods, the chapters on which contain much that is valuable and much that is useless; and while the police side of the question of food supply is fairly well covered, the hygienic side is very largely neglected and ignored. The chapters on air, water, and soil cover less than a hundred pages and are very sketchy; while the remaining chapters on habitations, lighting, and clothing, covering 50 pages, are of almost no value. The work is hardly a treatise on hygiene, for many of the most important subjects which make up that department of medical science are not touched upon at all. It is really a work on foods, far inferior to many written in our own language, with a few supplementary chapters on subjects which are much better handled in almost any modern work on hygiene.

C. H.

The Royal Medical and Chirurgical Society of London Centenary, 1805-1905. By Norman Moore, M.D., F.R.C.P., and Stephen Paget, F.R.C.P. The Aberdeen University Press, Limited, 1905.

The Royal Medical and Chirurgical Society of London is to be congratulated on the handsome volume in which it has commemorated its centenary anniversary. Its authors are both well known for their researches in the history of medicine, and their labor has evidently been one of love. The book will prove of value, not only as a history of the Society, but for the bibliographical and biographical information it contains concerning practically all the prominent physicians and surgeons who have flourished in London during the past century. There are also many interesting anecdotes and much curious and otherwise buried information about many medical worthies. It is sincerely to be hoped that a publication such as this will have a stimulating effect upon other medical organizations in the direction of preserving their histories and rendering information otherwise lost accessible to those who enjoy research in medical

history. Many interesting anecdotes could be quoted did space permit. There is an interesting account, for instance, in the sketch of Sir Henry Halford on the disinterment of the body of King Charles I., and in the biography of Elliotson it tells why Thackeray affectionately dedicated "Pendennis" to him, and of the disrepute into which he fell during the latter portion of his career. It is a pity that the circulation of a book such as this will, from the nature of its publication, be of so limited character. It could with profit be read by every member of the profession. F. R. P.

UNTERSUCHUNGS- UND BEHANDLUNGSMETHODEN KEHLKOPFKRANKHEITEN. VON DR. THEODOR HERYNG. Berlin: Verlag von Julius Springer, 1905.

PROF. HERYNG is one of the foremost laryngologists of the day, and the present volume will redound yet further to his honor. It may be regarded as an exposition of the most recent views in laryngology, and we know of no work in which they are presented in a more satisfactory manner. The work is divided into four sections. The first deals with methods of examination of the larynx and trachea. The second considers the non-operative treatment of diseases of the parts. The third section describes the various operative procedures practised within the larynx, and the last section deals with the instruments which are used in laryngeal work. There is also a very valuable section on the use of inhalations. One is particularly struck with the handsome illustrations with which the volume abounds. Another point which will make the book of especial value to the non-Teutonic reader is the clear and uncomplicated fashion in which the author presents his facts. The book is a distinct addition to the literature of the subject.

F. R. P.

SANATORIA FOR CONSUMPTIVES. A Critical and Detailed Description, together with an Exposition of the Open-air or Hygienic Treatment of Phthisis. By F. RUFENACHT WALTERS, M.D., M.R.C.P. With an Introduction by SIR RICHARD DOUGLAS POWELL, BART., M.D., F.R.C.P. Second edition. London: Swan Sonnenschein & Co., Ltd. New York: E. P. Dutton & Co., 1902.

To those, both lay and professional, interested in the great and growing subject of the prevention and cure of pulmonary tuberculosis, this book will be found to be invaluable in many ways. The author points out that consumptives, more than people in good health, differ in their powers of reaction, so that no one climate is

suited equally to all cases; that the localities which benefit the more curable cases possess certain essential features in common; and that where these conditions obtain it should be possible to conduct successful treatment. He asserts that neither high altitude, dryness, sunshine, fine weather, nor equable temperature should be regarded as essential, although useful or in certain cases desirable; that cures occur in the most unlikely climates; and that fresh air and medical supervision are more essential than fine climate; but climate must be carefully considered since certain climatic conditions are unfavorable to certain classes of patients. He presents a general review of the most modern methods of hygienic treatment; emphasizes the necessity of intelligent medical supervision of diet, accommodation, travelling, and recreation; discusses sites, grounds, construction, preparation, furnishing, and general sanitation of sanatoria, and the disinfection of sputa, linen, etc.; gives many interesting and valuable facts concerning results of sanatorium treatment, and presents a detailed description of the more important existing sanatoria in different parts of the world. The book is profusely and excellently illustrated. C. H.

MEDICAL PHILOLOGY. Gathered by L. M. GRIFFITHS, M.R.C.S. Eng. Part I., A–E 1. Bristol: J. W. Arrowsmith, 1905.

THIS little book comprises a series of notes on various obscure terms which are not applied in the popular language to medical conditions. We are all more or less familiar with the vulgar words which are used to indicate various portions of the human anatomy or applied to symptoms or ailments, but very few of us are familiar with their character or origin. The subject-matter of the book was published in the *Bristol Medico-Chirurgical Journal* when its author was serving in the capacity of assistant editor of that publication. Dr. Griffiths has rendered a distinct service to the medical profession in republishing them in book form. F. R. P.

PROGRESSIVE MEDICINE. Vol. II., June, 1905. A Quarterly Digest of Advances, Discoveries, and Improvements in the Medical and Surgical Sciences. Edited by HOBART AMORY HARE, M.D. Philadelphia and New York: Lea Brothers & Co.,

As usual this issue of *Progressive Medicine* contains a series of contributions of such varying character as to render it of importance to the whole profession.

The first is by Dr. William B. Coley and deals with hernia. A large amount of space is devoted to hernia occurring in infancy and childhood, a most important and not very well understood subject.

In the second part Dr. Edward Milton Foote presents the surgery of the abdomen, exclusive of hernia. The wonderful advances in this department of surgery render Dr. Foote's work particularly valuable. It is accompanied by an especially large series of fine illustrations.

Dr. John G. Clark contributes a valuable section on gynecology, including a particularly interesting discussion on the subject of spinal anæsthesia.

Stengel, in a lengthy but most important section, gives a thorough study of the recent progress made in diseases of the blood, diathetic and metabolic diseases, and diseases of the spleen, thyroid gland, and lymphatic system.

The volume is concluded by Dr. Jackson's valuable discussion of the recent progress in ophthalmology.

As is always the case, the book is not a mere compilation of the literature, but really presents a series of original articles on the progress of medicine. F. R. P.

THE PRACTICAL MEDICINE SERIES OF YEAR BOOKS. Comprising Ten Volumes on the Year's Progress in Medicine and Surgery. Issued monthly under the general editorial charge of GUSTAVUS P. HEAD, M.D., Professor of Laryngology and Rhinology, Chicago Post-Graduate Medical School. Volume II., General Surgery, edited by JOHN B. MURPHY, M.D., Professor of Surgery, Northwestern University Medical School. Series 1905. Chicago: The Year Book Publishers.

THIS volume of the *Practical Medicine Series of Year Books on General Surgery* gives a most complete *résumé* of the year's surgical literature. As the author states in the introduction he has tried wherever possible "to reproduce extensively a number of articles which treat in an analytical or statistical manner of the pathology or operations of an organ or set of organs, based usually on an enormous or extraordinary experience of an individual which was sufficient to make his statements authoritative." This plan, which the author has followed, we think a most excellent one, as much more can be learned from one article of this kind than from many reports of single cases from different individuals. Nearly one-half of the book is devoted to the "Surgery of the Abdomen," and this chapter would in itself make a most interesting and valuable book. The work as a whole is well written and is a most valuable reference book on recent surgical procedures. C. F. M.

THE HISTORICAL RELATIONS OF MEDICINE AND SURGERY TO THE END OF THE SIXTEENTH CENTURY. An Address Delivered at the St. Louis Congress in 1904. By T. CLIFFORD ALLBUTT, M.A., M.D. London: Macmillan & Co., Ltd., 1905.

PROF. ALLBUTT'S book will be read with pleasure by every physician who is at all interested in the foundation of his art. There are but few of us, unfortunately, whose familiarity with Latin and Greek permit of an intimate acquaintance with the works of the early masters of medicine and surgery, and, even though many of them are accessible in good translations, it is sad to record that the study of their works is almost entirely neglected by their successors. The labors of the distinguished author of this little volume should stimulate the flagging interest in antique and mediæval medical writings. Prof. Allbutt shows so much erudition and so close a familiarity with their work that, although the book is a small one it is evidently the result of years of study and labor. The author narrates the curious way in which medicine and surgery from time immemorial have at one time been closely associated, and at another drifted apart, and the alternation of glorious periods of advance with barren periods of dry rot is told in a delightful style. We are glad that the address is preserved in such a permanent and accessible form. F. R. P.

LA SCROFULE, TERRAIN SEPTICO-PYOHENICQUE HEREDITAIRE OU ACQUIS ET DIATHESE LYMPHOIDE PRE- OR POST-TUBERCULEUSE (MALADIE SYSTEMATISEE DU SYSTEME LYMPHATIQUE ET DE SES DEPENDANCES). Par M. LE DR. PAUL GASTOU, Ancien Chef de Clinique et Assistant de Consultation, Chef de Laboratorie de la Faculté à l'Hôpital Saint-Louis. Paris: O. Doin, éditeur, 1904.

IN this admirable monograph of more than 100 pages, Dr. Gastou sets forth, with great completeness, the modern conception of scrofula—a term which has passed through various vicissitudes of meaning, and for a time bid fair to disappear entirely from the nomenclature of medicine. According to the author's definition, scrofula is a septicopyæmia characterized clinically by the existence of hypodermites, or diffuse, prolonged, multiple suppurations, taking the form in one case of superficial impetiginous suppurations; in another of deep, ecthymatous or nodular abscesses, with engorgement, and sometimes suppuration of neighboring ganglia, and ending by the production of visceral alterations of the infection or sclerotic type, or of the degenerative type, such as fatty or amyloid degenerations.

The scope of the essay is readily gathered from the titles of its three parts: 1. Does scrofula exist, and how is it defined? 2. Refutation of the old conception of scrofula. 3. The modern scrofula.

The subject is one to which Dr. Gaston has devoted years of study, and in its present form his work must be taken as presenting the final word of the modern school, which took its origin in the early studies of Chaumier, published in 1881. Scrofula interests both the dermatologist and the pediatrician, and this monograph is, therefore, a welcome contribution to the current literature of both specialties, the value of which is greatly enhanced by the fine series of thirty-two half-tone reproductions and the four colored plates illustrating histological preparations. T. S. W.

A Text-book of Legal Medicine. By Frank Winthrop Draper, A.M., M.D. (Harvard), Professor of Legal Medicine in Harvard University; Medical Examiner for the County of Suffolk, Massachusetts; Medicolegal Pathologist at the Boston City Hospital; Fellow of the American Academy of Arts and Sciences, etc. W. B. Saunders & Co., 1905.

The term "legal medicine" has the same meaning as "medical jurisprudence," or "forensic medicine." As used by the author, it embraces a discussion of all branches of medical science as applied to the requirements of the law. The preface states that the book is intended chiefly for the use of students of medicolegal subjects, and as well for the practitioner. For the latter it can scarcely be of less value than for the former. It contains advice and instructions for the physician when called upon to appear as a witness, which every practitioner is likely to find opportune. Suggestions are made as to his demeanor, preparation, and rights, whether the proceeding be civil or criminal, whether he be called as an expert or as a witness to fact. Dr. Draper's experience as a medical witness renders him peculiarly qualified to advise.

The State of Massachusetts and several other New England States have abolished the office of coroner. The coroner or coronator is a very ancient officer of the law, and, as his title signifies, had originally to do with matters immediately concerning the Crown. It was required in the earlier days that he should be a knight and that he should maintain his office with fitting dignity. In addition to his familiar duty of holding inquests over dead bodies, his functions included preservation of the peace, inquiry into felonies, treasure trove, wreck, and, in emergencies, the execution of process.

The office has lost much of earlier importance in the communities where it survives. The inquest is the only prerogative now exercised, and, in the larger cities at least, it is conducted in such a manner as does not give great weight to the findings of a coroner's jury. It has been said that in our great metropolis the jury's verdicts are written up before the inquest is held, if it seems probable that questions may be raised concerning the liability of an influential corporation or individual. And it is stated that in other large cities the coroner's jury responds obligingly to the demands of the powers that control the respective municipal governments.

In Massachusetts a district medical examiner has succeeded the coroner. He is appointed by the Governor of the State, and is under bond for the faithful performance of his duties. All cases are now submitted to him, which would previously have been the subject of a coroner's investigation. He reports to a judge of the lowest grade in his district, who thereupon holds an inquest. The judge himself interrogates the witnesses and finds the facts. The subsequent proceedings are of the same nature as formerly.

The author has been medical examiner of the city of Boston, and is also professor of legal medicine at Harvard University. While, as he himself states, the work may be primarily intended for students of medical jurisprudence, his unusual practical experience, the fruits whereof are gathered into this book, gives it a wider scope, by reason of its interest to the medical examiner, the coroner's physician, and the practical doctor when summoned to appear in a legal proceeding.

The writer has evidently made a careful study of the authorities touching upon his various subjects, and to this has added his personal observations growing out of his official work and scientific research. The topics treated of cover a wide range—the identification of the living and of dead bodies, whether in a state of good preservation, decomposed, or mutilated. For that purpose inquiry is made into indications of sex, age, and physical characteristics. The questions of impotence and sterility are frequently of importance in their medicolegal relation, notably in cases of rape, bastardy, adultery, and divorce. The chapters on death from exposure to excessive heat or cold, hanging, drowning, strangulation, poisons, or noxious gases will possess an immediate interest for most of the readers, while the study of wounds, blood tests (to distinguish human blood), and of the medicolegal autopsy concern the coroner's physician or police surgeon more intimately.

As an example of the practical value of the book, a case may be taken where the physician is called upon to determine whether the crime of rape has been committed, by means of an examination of the victim or the perpetrator of the offence, or both. The physical examination may be considerably aided by the suggestions of the

author on this subject. But it must be borne in mind that the accusation of rape is easily made, and that the charge is, from its very nature, difficult to refute. It is frequently made falsely, although in good faith, by hysterical women, and often, too, deliberately and with malicious intent by others. The writer's observations concerning the manner of conducting the examination, the bearing of the accuser and the accused, and other surrounding circumstances may enable the examiner to be of great assistance in bringing about a just determination of the case. So, also, in questions arising out of criminal abortions or infanticide; these are points that come within the field of the physician's investigation other than those revealed by the physical scrutiny. The study of criminals and those upon whom injuries have been inflicted, for more than a quarter of a century, qualify the writer to speak with authority concerning such matters.

There is a chapter in which is discussed the legal status of the physician. Preliminary examination of those desiring to practice medicine is now required in most, if not all, the States of the Union.

This is followed by registration and a license to practice. The statutes of Massachusetts relating to this subject are taken as an illustration. The physician's liability to procedure, civil and criminal, is touched upon, and some of the leading cases are recited. The statutes of Massachusetts concerning the commitment of the insane, and as to the physician's duty to report births, cases of contagious diseases, and to issue certificates of death are probably not dissimilar from those enacted in other States.

It is to be regretted that Dr. Draper has not enlarged his volume to an extent that would permit him to give to the profession his views concerning the ethical relations of the physician to medico-legal matters, in a chapter specially devoted to the subject. More than once he condemns an association with a legal case whereby the physician undertakes any of the functions of a detective. This attitude will appeal to all reputable physicians. Yet in many cases, particularly those where the doctor's examination is the first, and yet more particularly where that examination is made by one in an official capacity, he may direct his attention to matters other than those that are purely medical. Thus, he should note the position in which a body is found, when there are indications of foul play; the direction from which the fatal blow may have come; spatterings of blood near the corpse; the general appearance of the surroundings, and the like.

It is the author's knowledge of such matters as these, coupled with a scientific treatment of his subject, that renders the volume timely and interesting. C. S.

ABDOMINAL PAIN; ITS CAUSES AND CLINICAL SIGNIFICANCE. By A. ERNEST MAYLARD, M.B., B.S. (Lond.), Surgeon to the Victoria Infirmary, Glasgow; Late Examiner in Surgery to the University of Glasgow and Victoria University, Manchester; Formerly Demonstrator of Anatomy Guy's Hospital, London; and Ex-President of the Glasgow Pathological and Clinical Society. London: J. & A. Churchill, 1905.

THIS book by Dr. Maylard is one of the most interesting and instructive books that has appeared lately in medical literature. The symptom abdominal pain confronts the physician more, perhaps, than any other symptom in abdominal disorders, and therefore a book on this subject cannot be other than valuable to all medical men. The first part of the book is taken up with description of the nerve supply of the abdominal wall and viscera, and four diagrams illustrate the various cutaneous areas of referred pain in abdominal diseases. The causes, varieties of pain, also the diagnostic differences of pain arising from diseases of the various organs are gone over at length. The last part of the book is devoted to the surgery of the abdomen, the different incisions for opening the cavity are described and illustrated by a diagram; also methods for closing are mentioned. The treatment of a patient before and after laparotomy is given, and some space is allotted to the results of abdominal operations. The book is well written and cannot be too highly recommended for its practical usefulness. C. F. M.

SAUNDERS' POCKET MEDICAL FORMULARY. By WILLIAM M. POWELL, M.D. With an Appendix containing Posological Table, Formulæ, and Doses for Hypodermic Medication, Poisons and their Antidotes, etc. Seventh edition, revised. Philadelphia and London: W. B. Saunders & Co., 1905.

THIS handy little volume has amply proved its usefulness by the great favor with which it has been received. The first edition was issued in 1892. It has been through the subsequent years revised from time to time and kept thoroughly up to date. In addition to the formulæ, posological tables, etc., it contains the usual tables of poisons and the formulæ of obstetrical data, incompatibles, etc. There is also a very unique little surgical remembrancer, said to be from the pen of an eminent London surgeon, and containing many invaluable hints which every practitioner would do well to learn and remember. There are also some very excellent lists and hints of antiseptic surgery. The book is one of the best of its class. F. R. P.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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The Fourth Disease of Dukes.—V. BOKAY (*Deutsche med. Wochenschrift*, 1904, p. 1651). In July, 1900, Dukes published in the *Lancet* an article entitled "On the Confusion of Two Different Diseases under the Name of Rubella," and designated an infection which he thought could be distinguished by definite characters "fourth disease." This group of cases bears the same relation to scarlet fever that true rötheln does to measles, mild cases of measles being easily confused with rötheln and abortive cases of scarlet fever with the fourth disease. Dukes bases his claim upon the following observations: In 1892 he was called by a director of a school to see 16 boys ill, presumably, with scarlet fever. Having been for some years convinced of the existence of a separate disease confused with mild attacks of scarlet fever, he recognized this type in all the cases, and, although the boys were isolated but fourteen days, not a single instance of transmission of the disease followed their return to their families. In another school epidemic he found, among 31 patients, cases of both scarlet fever and the fourth disease, but, while infection in the former followed in from two to three days after exposure, the incubation period in the latter was prolonged to fourteen or fifteen days. Nine of the children with the fourth disease developed later typical scarlet fever, and 1 case of scarlet fever developed later fourth disease. One of the children with the fourth disease had previously had scarlet fever, and a number of them had rötheln. In a third epidemic of 19 cases of the fourth disease, 42 per cent. of the patients had previously had rötheln.

From these observations, he draws a clinical picture of the disease. The incubation period varies from nine to twenty-one days, and while there are in some instances mild prodromal symptoms, as a rule, the eruption is the first evidence of the disease. This eruption is of a pale, red color, with a finely and compactly papular appearance, scarcely raised above the level of the skin. In a few hours it covers the whole

body, being less evident on the face, while an area about the lips and nose is usually spared. The pharynx is reddened, the tongue coated, and the conjunctivæ injected. Glands in the neck are little swollen, firm, not so large as in rotheln. The eruption fades rapidly and is followed by a general scaly desquamation, which is complete in from one to two weeks. Renal complications never follow. The general health is only slightly affected. The pulse in mild cases is scarcely increased in frequency. The temperature varies from 98.4° to 104°. Constitutional symptoms disappear when the rash is well out, and the child can be out of bed in five or six days. Isolation may be safely discontinued after two or three weeks.

Although Dukes' publication gave rise to a lively polemic among English and American authors, the article of Weaver in the *Journal of State Medicine*, 1901, is the only extensive subsequent contribution to the subject. As medical superintendent of a hospital for infectious diseases, he has been impressed with the frequent occurrence of apparent relapse in scarlet fever characterized by the reappearance of fever and of an eruption. Dukes' article offered an explanation for these manifestations, and on studying the cases carefully, he was able to corroborate Dukes' report in all but minor details.

General acceptance, however, has not been accorded these views, which many prominent authorities ignore, while others oppose them. v. Bokay, from his own experience, feels sure that all who deal much with the infectious diseases of children have met with instances similar to those described by Dukes and Weaver. He believes that he has frequently encountered the disease, but has no observations to cite which would be conclusive enough to strengthen its position. In a review of the literature, however, he finds several important contributions to this question prior to the publications of Dukes. Filatow referred to it in 1885 and devotes a chapter to its discussion under the title "Rubeola Scarlatinosa," in his book on the *Infectious Diseases of Childhood*, published in 1896. The main observation upon which he based his description was this: In a family of eleven children from one to sixteen years of age, scarlet fever was introduced in 1882, three children being attacked. Two years later one of the boys developed, during convalescence from influenza, a profuse, densely punctate, scarlatinoid eruption without angina and with but slight fever. After two days another child developed an identical eruption; on the third day two more children were affected, and just after these the two oldest. The constitutional symptoms were very mild in all cases, and a governess who had previously had scarlet fever was also attacked. Considering these instances of abortive scarlet fever, Filatow assured the family they need have no fear of future outbreaks of the disease. In October, 1885, much to his surprise, scarlet fever again broke out in the family, and four of the children who had previously had the mild eruption became ill, one dying. Filatow defines his position thus: "Under the name of rubeola scarlatinosa I designate a specific, acute, infectious, and contagious disease characterized by a scarlatinoid eruption, but distinguished from scarlet fever by its uniformly mild course and principally by the distinct features of its virus."

Postoperative Parotitis.—WAGNER (*Wiener klin. Wochenschrift*, 1904, Band xvii. p. 1407). Munde in 1878 described the first case of postoperative parotitis following an ovariotomy. In the succeeding years

many more instances were added, and the view which at first prevailed that it was a complication peculiar to operations upon the ovary was soon disproved. In a careful and exhaustive review of the literature, Wagner accepts only 38 cases as coming without doubt into this group, and adds 5 more from Eiselburg's clinic. A composite clinical picture is as follows: In from two to fourteen days, usually from the fifth to the seventh day after operation, the parotid on one side begins suddenly to swell, accompanied by a rise in temperature; this is usually soon followed by a swelling on the other side. The gland continues to enlarge and becomes very painful, hard, and tender, and is surrounded by an area of marked œdema. Pain on opening the mouth and dryness make swallowing almost impossible. At this stage a few of the cases subside and the swelling recedes, but most go on to suppuration, and if not incised opportunely, may rupture into the auditory canal. Pus is not always obtained on incision; there may be diffuse infiltration, with partial necrosis of the gland. The complication is a serious one, averaging in the 42 cases a mortality of 30.2 per cent. Death usually occurs with pyæmic manifestations.

In Wagner's 5 cases the patients were all but 1 over fifty years of age and 4 of them much emaciated by their illness. The diseases and operations were first gastric ulcer, gastroenterostomy second, carcinoma of œsophagus, gastrostomy; third, carcinoma of colon, resection of cæcum, ascending and transverse colon; fourth, carcinoma of pylorus, gastroenterostomy; fifth, cholelithiasis, cholecystectomy. The duration of operation was in 1 case two and a half hours; in 2, one and a half hours, and in the remaining fifty and sixty minutes respectively. In all of the cases vomiting occurred after operation. The parotitis developed on the second, fifth, sixth, tenth, and fourteenth days after operation. In all but 1 case the temperature remained normal up to onset of the complication, and in this 1 there was merely a slight and temporary rise on the day after operation. In 1 instance the complication ran an afebrile course; in 1 there was only slight fever; in another the temperature was normal until shortly before death; while in the other 2 there was a rise to 38.9° and 40.2°. In 3 cases the swelling went on to suppuration, and all 3 died of a terminal bronchitis and lobular pneumonia. In 1 of the cases the submaxillary gland alone was involved; in the other 4 the parotid, twice unilaterally, twice bilaterally.

Wagner thinks the weight of evidence is in favor of mouth infection rather than by the way of the blood current. The histological evidence advanced by Hanson is almost conclusive. The secondary parotidides in infectious diseases are usually caused by organisms commonly found in the mouth and differing from those causing the original disease. In pyæmic processes, moreover, the parotids are seldom implicated. As has been noted, the earlier authors considered the parotitis to depend primarily upon some injury to the ovaries, and, although cases are reported after operation in other fields, the largest number have followed pelvic manipulation. The intimate relation between the genital and salivary glands is well illustrated in mumps; while injury to the testicles is often followed by swelling of the parotids, and many interesting cases are on record of marked functional derangement of the glands in the course of ovarian disease. Skeene-Keith is the only observer who considers the apparent relation between the two purely a matter of coincidence. Pawlow has shown that opening the abdomen and

handling the intestinal coils cause a marked diminution in the flow of the submaxillary secretion. v. Preuschen, on the basis of these experiments, urges that the laparotomy itself, especially when prolonged, is the essential factor, the slowing of secretion allowing more readily an entrance of organisms from the mouth. Berth lays emphasis upon the paralysis of the salivary nerves by the narcotic. Cases of parotitis follow operations other than pelvic, even when the abdominal cavity is not opened. Chloroform paralyzes the salivary nerves. Ether stimulates them, but stimulation is followed by a period of diminished salivary secretion. All of these factors would favor infection from the mouth. Wagner believes trauma by the anæsthetist to be of importance, as experiments show that the parotids can be infected from the ducts only after injury.

An Instance of Family Tabes arising on a Syphilitic Basis.—NONNE (*Berliner klin. Wochenschrift*, 1904, No. 32, p. 345) adds another interesting instance to the growing list of family tabes depending on syphilitic infection. A girl, aged twenty years, was admitted to the hospital complaining of a sense of uncertainty upon her legs, shooting pains, and loss of vision. After a normal birth and childhood she began, at ten years, to gradually lose her eyesight, and for two years prior to her entrance had been almost totally amaurotic. The left eye squints since her thirteenth year. Locomotive uncertainty began in the tenth year, and a little later the paroxysmal pains in the legs. The poorly nourished, rather pale girl showed double optic nerve atrophy, unequal, immobile pupils, old opacities in left cornea, slight static and locomotor ataxia, positive Romberg's symptoms, hyperalgesia, and delayed pain sensation on feet and legs, hyperæsthesia to cold over back, and absence of the patellar and Achilles reflexes. There was no superficial evidence of syphilitic disease and no history of acquired infection. The mother of the girl suffered for twenty years with dysuria, occasional severe lancinating pains, and some unsteadiness on the legs. Her first child was born prematurely at eight months and died a few weeks later with an eruption called smallpox; the second was also born prematurely and died of general weakness; the third and fourth are alive and healthy; the fifth pregnancy ended in an abortion, and four years later the present patient was born. Examination of the mother showed inequality of the pupils, with immobility to light and sluggish reaction on accommodation, absent patellar and Achilles reflexes, slight but definite ataxia in both legs, positive Romberg's symptom, insular hyperalgesia, and delayed conduction on feet and legs, and hyperæsthesia to cold on abdomen. No evidence of previous syphilis could be obtained. Further examination was made of a brother and sister. The brother, aged twenty-five years, was perfectly normal in every particular. The twenty-nine-year-old sister had been married five years and had had two healthy children. She, too, had suffered for sixteen years from lancinating pains and weakness in the legs, and for seven or eight years her sight had been failing. Examination revealed: Argyll-Robertson pupils, Achilles reflex absent on both sides, and patellar on one, slight ataxia and hyperalgesia of lower extremities. In the eyes there was an old choroiditis and retinal opacities. The ophthalmologist Beselin regarded the eye lesions in both sisters as of luetic origin.

Nonne observes that this instance indicates the peculiarities of hered-

itary lues pointed out by Marburg, namely, that it affects women more frequently, and that eye symptoms are prominent and ataxia slight. Both daughters showed definite stigmata of inherited syphilis. Another point of interest is that the disease spared the brother who was born between the two sisters. Why the nervous system should be so prominently attacked in certain cases and spared in others remains undetermined. The influence of surroundings of a family disposition and the peculiarities of different varieties of syphilitic toxin have been urged. Nonne cites instances which apparently support each of these. A man and woman previous to marriage had both independently acquired syphilis; later both developed basal meningitis. Two brothers living apart and infected from different sources, both became tabetic. Before marriage a woman had contracted syphilis from a man with whom she later never lived; both developed tabes.

SURGERY.

UNDER THE CHARGE OF

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Ectopy of the Testicle.—LANZ (*Zentralblatt f. Chirurgie*, April 22, 1905, p. 425) says that although ectopy of the testicle refers generally to every testicle which does not occupy its normal place in the scrotum, we distinguish between ectopy of the testicle, which includes every testicle that deviates from its normal path to the scrotum, and retention of the testicle, in which the organ is held somewhere in its normal path of descent. Retention is much the more frequent.

The statement of Curling that a testicle that does not descend within the first year of life will remain permanently ectopic has been widely disputed. Lanz saw a case in which at puberty the testicle first appeared at the external inguinal ring and finally reached the neck of the scrotum.

Although the cause of the trouble is not known, an hereditary tendency is certain. Lanz saw it occur in two brothers. He has never found anything by operation to explain it. In a total of 51 operations he has never found the strong peritoneal adhesions which would result from a recovered case of fetal peritonitis. The final descent in a case of retention is never prevented by the vas deferens, although the shortness of the vessels occasionally interfere.

Almost without exception the testicles show striking macroscopic changes. They are smaller, flabbier, often entirely atrophied, and the always well-developed epididymis is lifted up from the testicle. Since

the nerve and blood supply are no more deficient than when the testicle is in the scrotum, this is not a cause. It is probable that the macroscopic deficiency of the organ, resulting from pressure, a failure of development as such, is the cause of the failure of descent.

In 11 of his 51 cases one-sided castration was done, on account of the marked atrophy and the uncertainty of a safe descent later.

In only 1 case could active spermatogenesis be shown. The albuginea was without exception thickened, in a few cases three to five times the normal thickness. The interstitial connective tissue was sometimes scarce, sometimes much increased, so that the seminiferous tubules were scattered about as isolated islands. Of especial interest were some collections of atypical glandular epithelium, which seemed to support Cohnheim's theory concerning the roll played by misplaced embryonal cells. This is of especial value in relation to the testicle, in which it has been claimed that malignant degeneration occurs earlier than usual. This has lately been disputed. Lanz's findings tend to show an especial inclination of the testicle to malignant degeneration.

Patients usually seek help, not on account of the retention of the testicle, but of the associated complications, the most frequent of which is hernia. Lanz found that the patients rarely complained of sexual disturbances, while, according to the literature, this seems to be very frequent, indeed, the monorchid has been compared to a castrated person. He advises castration when the testicle is markedly atrophied. It prevents a return of the condition, while orchidopexy is frequently followed by return, and it prevents later malignant degeneration. According to his experience, the remaining testicle will, by enlargement, tend to assume the duty of the removed organ.

Of 14 cases treated in the last two years, in 6, or nearly half, castration was done. Only exceptionally were children in the first decade operated on. Massage was advised instead—*i. e.*, stroking along the inguinal canal with a drawing down of the testicle. In 8 of the 14 he did an operation which he calls an extension of the testicle. After dividing the anterior wall of the inguinal canal, he opened the processus vaginalis, and made the testicle present at the external ring. He cut the vaginal process and treated the upper portion as a hernial sac. He then stretched the spermatic cord carefully and reconstructed the posterior wall of the canal on the Bassini principle. Then with a long suture he caught the testicle near its lower pole and passed the suture through a button-hole in the fundus of the scrotum, often poorly developed. Under tension the suture is fastened, with strips of plaster, to the adjacent part of the thigh, or it is bound to a transverse bar fastened between the slightly spread thighs by plaster-of-Paris bandages. The inguinal wound, as well as the button-hole, are then closed.

An Experimental and Histological Study of Cargile Membrane.—CRAIG and ELLIS (*Annals of Surgery*, June, 1905, p. 801) made a very interesting series of experiments upon dogs to determine the value of Cargile membrane in preventing adhesions after operation, especially in the peritoneal cavity. Its irritative action seemed to be the principal factor militating against the otherwise beneficient possibilities of the material.

Whether chromicized or unchromicized, the Cargile membrane is destroyed and disappears, the former in no case lasting to macroscopic view longer than fourteen days, the latter five days. Its presence micro-

scopically was noted some time later. The two doubtless bear relatively the same relation to each other as do the same varieties of catgut.

Neither variety can be depended on to remain where placed. In a situation subject to peristaltic activity the membrane must be anchored, as by sutures. Since both act as irritants, they are of no value in preventing adhesions within the peritoneal cavity.

The membrane is of value in preventing adhesions to wounded nerves and tendons, when such structures lie in tissues subjected to trauma, operative or otherwise. The chromicized is the more valuable. Several layers make a safer and better protection than one layer.

Used to replace destroyed or removed dura mater, the unchromicized is exceedingly difficult to handle, and would be of no special value. Owing to the facility with which the chromicized variety can be handled, its greater toughness and increased power to resist absorption, it would prove of greater value in replacing the dura.

These studies indicate that the membrane is destroyed by a lytic substance or substances contained in the body fluid. Phagocytosis may be safely excluded as a chief important cause.

The Diagnosis and Treatment of Fracture of the Carpal Scaphoid and Dislocation of the Semilunar Bone, with a Report of Thirty Cases.—Codman and Chase (*Annals of Surgery*, March and June, 1905), in an extensive paper, consider that the profession at large does not realize that many sprained wrists are instances of fracture or dislocation of the carpal bones, and they believe that the treatment should be as definitely known as those of fracture or dislocation of any other joint. They summarize the important points as follows:

1. Sprains of the wrist which do not promptly recover are in many cases fractures or dislocations of the carpal bones.

2. The large majority of such carpal injuries are either simple fractures of the scaphoid or anterior dislocations of the semilunar bone.

3. These two injuries are frequently combined, and in such cases the proximal fragment of the scaphoid is usually dislocated forward with the semilunar.

4. Simple fracture of the scaphoid gives a definite clinical picture and may be recognized, even without the *x*-ray, by the association of the following symptoms, viz.: *a*. The history of a fall on the extended hand. *b*. Localized swelling in the radial half of the wrist-joint. *c*. Acute tenderness in the anatomical snuff-box when the hand is adducted. *d*. Limitation of extension by muscular spasm, the overcoming of which by force causes unbearable pain.

5. A broken scaphoid has little power of repair and appears capable of but little callus formation.

6. Fractures of the scaphoid which remain untreated or are treated by massage and active and passive motion generally if not always remain ununited, and the original symptoms persist for years with only slightly abated intensity.

7. Cases of fracture of the scaphoid may unite, if motion of the wrist is prevented during the first four weeks after the injury, but if by this time no union has occurred, future union is unlikely.

8. Excision of the proximal half of a fractured scaphoid gives a somewhat better result than conservative treatment.

9. A posterior incision to the outer side of the tendons of the extensor communis digitorum gives an easy and safe access to the proximal half of the scaphoid.

10. Passive motion of the wrist-joint and active motion of the fingers should be begun within a week after this operation.

11. The possibility of a bipartate scaphoid should be considered in interpreting *x*-rays of simple fractures of the scaphoid, but its occurrence must be very rare in comparison with fracture.

12. Anterior dislocation of the semilunar bone should be recognized clinically, even without the *x*-ray, by the association of the following symptoms, viz.: *a*. The history of an injury of considerable violence to the extended or twisted wrist. *b*. A silver-forked deformity, the posterior prominence of which corresponds with the head of the os magnum, and between which and the lower end of the radius is found a groove representing the position formerly occupied by the now anteriorly dislocated semilunar. *c*. A tumor under the flexor tendons of the wrist just anterior to the lower end of the radius. *d*. A shortened appearance of the palm as compared to the other hand. *e*. Stiffness of the partially flexed fingers, motion of which, either active or passive, is painful. *f*. The persistence of the normal relations of the styloid processes of the radius and ulna and the existence of shortening of the distance from the radial styloid to the base of the first metacarpal.

13. Recent dislocations of the semilunar may be reduced with good result, even after the fifth week, by hyperextension followed by hyperflexion over the thumbs of an assistant held firmly in the flexure of the wrist on the semilunar.

14. Irreducible dislocations demand excision of the semilunar and the whole or a portion of the scaphoid, if there is a coincident fracture of the latter.

Gastroenterostomy and Pyloroplasty: An Experimental Study by Means of the X-ray.—CANNON and BLAKE (*Annals of Surgery*, May, 1905, p. 686) experimented on cats. Two kinds of food were used, thin-boiled starch to test the passage of fluid contents from the stomach, and canned salmon as a more natural food. Both were mixed with a small amount of subnitrate of bismuth, so that the course of the food could be easily watched by means of a fluorescent screen.

They concluded that the stomach is not a passive bag. During digestion the cardiac end slowly contracts, while over the pyloric end peristaltic waves are constantly running, churning the food with the gastric juices and forcing the chyme into the intestine. Observations on the human stomach show that as the stomach empties the pylorus becomes the lowest point, so that even if gravity drainage occurred the pylorus is the natural outlet as long as the stomach retains the power of contracting.

Intra-abdominal pressure is approximately atmospheric, and the pressure in any part of the passive alimentary canal depends on the weight of overlying abdominal organs. If the canal is inactive, the food is, therefore, as if surrounded by water. Gravity cannot act, and gravity drainage does not occur.

Because the peristaltic waves move toward the pylorus, the intragastric pressure is greatest there, being three or four times greater than at the cardiac end. Unless a gastroenterostomy opening or stoma is

close to the pylorus, the food, even when fluid, is pushed through the pylorus rather than the stoma.

Circulation of the food through the pylorus to the duodenum and back to the stomach through the anastomosis was repeatedly observed, but it was not followed by the clinical symptoms of a "vicious circle." The circulation of food was observed best when the stomach was full, the intestinal wall, by stretching of the stomach, being drawn up into line with the gastric wall and closing the opening. The symptoms usually ascribed to a "vicious circle" were shown to be associated with a kink of the intestine just distal to the anastomosis. This is due to a break in the peristaltic wave from the division of the circular muscular fibres of the bowel at the opening.

Food passing through the stoma from the stomach is highly acid and is not brought into contact with the neutralizing juices of the duodenum. Ulcers which occur occasionally in the jejunum, opposite or near the stoma, may be due to the abnormal presence there of acid chyme. No case of ulcer of the duodenum after pyloroplasty has been reported.

The stoma should be large and as near the pylorus as possible, and overeating should be avoided to prevent the closure of the stoma by stretching of the stomach. Kinks may be oviated best by attaching a narrow band of the distal gut to the stomach for several centimetres beyond the stoma, thus permitting peristalsis to become an effective force. The probability of a "vicious circle," the non-mixture of food with the duodenal juices and the dangers from kinks in gastroenterostomy make the operation not an ideal one. In pyloroplasty these objections are avoided.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Serum Treatment of Exophthalmic Goitre.—DR. K. THIENGER states that Moebius has had good results with the serum of sheep from which the thyroid glands had been extracted six weeks before bleeding. This serum was given by mouth in wine. Following its administration the goitre diminished and the constitutional symptoms were ameliorated, but the condition of the pulse remained unchanged. The author has treated 4 patients with this serum. During the treatment no other medication was given, save bromides, to one of the cases. The general condition was improved by the treatment, the pulse rate

became slower, but in 3 of the patients the size of the goitre was unchanged, and the exophthalmos persisted. Possibly, in the author's opinion, these facts may have been due to the small dosage employed. A remarkable result, however, was achieved in a man, aged twenty years, with typical exophthalmic goitre, who was in very bad condition when first seen, presenting all the symptoms of a profound toxæmia. Four days after the institution of the serum treatment the size of the goitre was lessened, the pulse rate had fallen from 140 to 98, and the improvement continued until seven weeks later the patient was discharged with very little exophthalmos, an increase in weight, and a pulse rate of 76.—*Münchener medizinische Wochenschrift*, 1905, No. 1, p. 15.

Atropin in Puerperal Fever.—Dr. R. Drenkhahn has used this drug for ten years in cases of parturition or abortion followed by rise in temperature, and regards it as a useful agent in this connection. It is superior to opium, in that it does not cause constipation, and in that the pupil gives an index of its effect. Atropine is efficacious only when the infection is confined to the uterus and is incapable of influencing an infection which has involved the lymphatics and has become generalized. The author reports a case in a primipara, aged twenty-six years, with the infection confined to the uterus, in which complete cure in nine days followed the exhibition of atropine. Another patient in whom the infection had become generalized was unaffected by the drug. The reason for the action of atropine in this condition lies in the paralytic action which it exercises upon the uterine muscular fibres.—*La semaine médicale*, 1905, No. 7, p. 78.

Serum Therapy in Syphilis.—Dr. F. J. Bosc, impressed by the similarity between sheeppox and syphilis, has prepared a serum from the blood of sheep previously injected with blood from syphilitic individuals. Having assured himself of the harmlessness of this serum, he has injected it into patients, both before and after the appearance of the eruption. In the former class of patients the rash was not, as a rule, prevented from appearing, but it consisted merely of macules and was delayed beyond the usual time. One patient injected on the eighteenth day after the appearance of the chancre, showed no eruption two months later. The dose of the serum was 5 drachms.—*La semaine médicale*, 1905, No. 2, p. 20.

Methyl Alcohol.—Dr. H. W. Wiley, in a paper upon this substance and its uses, states that it is probable that its toxicity is due, in considerable measure, to impurities, but, nevertheless, he considers that its addition, either pure or refined, to beverages or medicines cannot be too severely condemned. He also believes in its elimination from liquids for external use, such as bay rum, and considers that it would be wise to lay a small tax upon wood alcohol, both in manufacture and sale, for the purpose of establishing over it the same legislative supervision which attaches to ethyl alcohol, and which supervision should extend, as it ought to do in the case of grain alcohol, to all remedies and beverages made therefrom. All medicines and remedies containing alcohol, which are not official, should be subjected to the same regulations in manufacture and sale as the corresponding quantity of alcohol

sold as such. The sale of medicines of which alcohol is the chief valuable constituent, without control, without notification, and without the knowledge of the consumer, is a condemnable practice, whether the alcohol be ethyl or methyl. If it be the latter, the practice should receive additional condemnation, because of the greater toxicity of this compound.—*American Journal of Pharmacy*, 1905, No. 3, p. 101.

Mercury Lactate in Syphilis.—Dr. A. Bouveyron prescribes, in the early stages of syphilis and later, when the buccal or lingual mucous membranes are affected, pastilles containing 0.075 grain of mercury lactate, which the patient is told to dissolve in the mouth after eating. The dosage is six per day. The pastilles have very little metallic taste, and they have a distinct effect in buccolingual syphilides; the mucous patches become less painful and tend to heal quickly. Syphilides of the throat are less favorably affected, although dysphagia becomes less distressing. The pastilles also act well in mercurial stomatitis, because of their bactericidal effect upon the micro-organisms of the mouth. Their only disadvantage is that they blacken the dorsum of the tongue.—*La semaine médicale*, 1905, No. 3, p. 30.

Veronal Poisoning.—Dr. H. Kress has observed, in 3 neurasthenic patients who had taken 7½ grains of veronal nightly for insomnia, symptoms of intolerance after three or four days, characterized by marked somnolence, loss of appetite, and weakness. Dr. J. Kuhn has observed a patient who had taken the drug in the same dosage, and after a few days exhibited a slight febrile movement, with dryness of the mouth and burning sensation in the throat. A morbilliform eruption appeared upon the face, which, in a few days, extended to the chest and arms and became confluent; this was followed by a vesicular and bullous eruption upon the buccal and pharyngeal mucous membranes, conjunctivitis, and pain in the ears. For six days the elevation of the temperature persisted, and not until the eighth day did the symptoms disappear. From these cases we may conclude that veronal may exercise a cumulative action and cause toxic symptoms more or less grave.—*La semaine médicale*, 1905, No. 6, p. 69.

The X-rays and Sterility.—Drs. F. Tilden Brown and Alfred T. Osgood report upon the *x*-ray as a factor in the causation of sterility. Eighteen instances among *x*-ray workers have been observed by them, and of these all who had done extensive work with the ray for more than three years showed absence of spermatozoa in the seminal fluid, while those who had been engaged in the work for a shorter time and had been careful to avoid direct exposure showed different states of oligonecrospermia. Other cases were examined whose exposures have been infrequent and short, whose seminal fluid was normal. The cases showing sterility are men in excellent health. There has not been the least sign, subjective or objective, of the development of the sterility, and there has been no deterioration of the sexual activity. One-half of the men are married, and none of these has had a child since he undertook this work. The authors cite an instance which had been treated by the *x*-rays for pruritus ani, with the result that, notwithstanding the use of a shield, a necrospermia was produced. This cleared up in five months. In the more thoroughly exposed cases, however, the question of prognosis

must remain in abeyance until evidence of the recuperative power of the seminiferous tubules be collected. Meanwhile the operator of Roentgen ray tubes should work behind a screen impenetrable by the rays, and should expose himself as little as possible. Proper tube shields should be used. The authors conclude with the statement that repeated, prolonged exposures of the testes causes sterility in the human being. Adequate protection to all parts of the body not directly exposed for examination should invariably be provided.—*American Journal of Surgery*, 1905, No. 9, p. 179.

Renal Organotherapy.—Dr. F. Choupin reports results from the administration of macerated pork kidney. A uræmic patient, over whose symptoms ordinary treatment had no influence, was greatly improved as soon as he began to take 10 drachms of the kidney daily. In another patient the substance was given for seven months. On suspension the uræmic symptoms reappeared in about fifteen days, but disappeared upon resumption of the treatment. This form of treatment has certain drawbacks, such as gastric disturbance, skin eruptions, etc., but these are never grave, and disappear soon after the suspension of treatment. The skin eruptions seem to signify that the medication is acting favorably. The kidney substance has a marked diuretic action, diminishes hypertension of the arteries, and also exercises a certain antitoxic effect. Its influence over albuminuria is *nil*, yet it causes amelioration of all symptoms. The author uses the kidney macerated without previous washing to remove the urine, and adds to it syrup of limes; he considers two kidneys daily the proper dosage.—*Revue de médecine*, 1905, Nos. 1 and 2, pp. 64 and 130.

Dechloridation in Phlegmasia Alba Dolens.—Dr. H. Signoret has employed this means in post-partum phlegmasia. In his opinion, the treatment is applicable of other forms of the affection, especially those accompanied by œdema. Before beginning the treatment, it is necessary to determine the degree of retention of chlorides. To diminish these we need only remove the salt from the diet, both that used before and after cooking. The diet itself is of little importance, the preference being given to broiled meats, vegetable purees, cooked fruits, and pastry. The progress of the treatment is best tested by estimation of the chlorides excreted by the urine, frequent weighing of the patient being contraindicated by reason of the possibility of causing embolism. In 6 patients observed by the author, the œdema disappeared in a comparatively short time, ten or twelve days at most, and the dietetic changes were well borne by the patients.—*La semaine médicale*, 1905, No. 8, p. 91.

Treatment of Hay Fever.—Prof. Alfred Denker, while acknowledging the advances in the treatment due to the elaboration by Dunbar of his antitoxin, which is extracted from the pollen of graminaceous plants, advocates what is, in his opinion, a preferable method which acts favorably, not only upon the attack, but also prevents recurrences, at times for two years. To allay the irritability of the nasal mucosa, the author resorts to massage of this stricture after preliminary anæsthesia, which is brought about by means of an application of a solution of cocaine hydrochlorate, 10 parts; adrenalin, 0.1 part; distilled water, 100

parts. With this the entire nasal lining is swabbed by means of cotton upon an applicator, or if the patient is very sensitive, a spray of 1 per cent. cocaine may be employed first. After the anæsthetic has taken effect the lining of the nose is massaged by means of a cotton swab dipped in a 10 per cent. of oily solution of europhen, the pressure used being gradually increased. The procedure is carried out daily for from two to four minutes for each nasal cavity, depending upon the sensitivity of the patient. In cases with marked conjunctival involvement a few drops of tincture of opium and water, equal parts, are instilled. The treatment is effective in preventing a recurrence during the following season in the majority of cases.—*Münchener medizinische Wochenschrift*, 1905, No. 19, p. 902.

PEDIATRICS.

UNDER THE CHARGE OF

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AND

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Thymic Stridor among Infants.—C. HOCHSINGER (*Revue mensuelle des maladies de l'enfance*, 1905, vol. xxiii., No. 3, p. 97) examined 58 infants with the radiograph from the standpoint of the size of the thymus. He found an enlarged thymus in 26 of them, and 20 of these presented the symptom-complex of congenital stridor. He considers this disease due to a compression of the trachea induced by the enlarged thymus gland. In addition to the radiographic examination, he considers percussion of the thorax as important in making a diagnosis; he further refers to the connection between enlargement of the thymus and hereditary syphilis, as well as that existing between hypertrophy of the spleen and the thymus.

Treatment of Capillary Bronchitis with Mustard-water Pack.—O. HEUBNER (*Therapie der Gegenwart*, 1905, Band xix. No. 1) has employed this method for several decades, fighting with it particularly the capillary bronchitis of young children up to the third year, and particularly those acute cases of mucopurulent catarrh leading to danger of asphyxia within a few hours or days; they occur especially often in the course of measles and whooping-cough, but sometimes also without apparent infection; the greater number of the patients are weak children with a rachitic thorax. They are usually very much prostrated, their faces deadly pale, the respiratory frequence, however, very high. A number of 70 to 80 respirations is not out of the ordinary, the pulse is small and frequent, the fever high. The thorax is filled with many very fine rales, showing that the greater part of the bronchial tree is filled with tough mucopus. The treatment consists in mixing 1 pound of mustard flour with 3 pints of hot water; the mixing process is continued for a matter

of ten minutes—that is, until the oil from the mass irritates the nose and eyes. A large sheet is now saturated with the mustard solution, wrung out and spread over a woollen blanket. The entire body of the patient from the neck down is rolled in the covers, which are tied about the neck and feet, so that the child will not have to breathe the mustard-laden air. After from ten to twenty minutes (depending upon reaction and strength of the patient) the child is taken out; its skin will be a lobster red; a hot bath or sponge comes next, and the child is then wrapped in a warm-water pack, in which it remains from one to two hours; if the child's head becomes very red or marked perspiration sets in, the cloth is at once unrolled, and a second warm bath, followed by a cooling shower, is resorted to. This ends the procedure for the day. Within twenty-four or forty-eight hours the patient is usually much better, and convalescence is established very soon after.

A Foreign Body in the Larynx of a Boy.—HOEDLMOSER (*Wiener klin. Wochenschrift, 1905, Band xviii., No. 13, p. 332*) relates the case of a boy, aged nine years, who, while eating soup six days before his admission to the hospital, swallowed a piece of bone; as it gave him no trouble the first day after the accident, it was thought to have reached the stomach; on the second day he had slight difficulty in breathing, and an examination showed the bone fastened in the larynx. Breathing became more and more difficult until the day of admission, when a violent respiratory stridor, especially on deep inspiration, was noticed. The laryngeal examination revealed a swollen and reddened mucous membrane; in the sagittal direction of the larynx a white furrow could be made out, passing from the anterior to the posterior wall; on each side of it a small lumen could be seen. Extraction of the foreign body was successful on the fifth attempt; no cocaine was applied because of the very marked dyspnœa. The bone was found to be triangular; its flattened surface 2 mm. thick, its sides 17, 17 and 18 mm. long. The remarkable part of the case is the long time the bone remained in the larynx, the successful endolaryngeal removal at one sitting, in spite of the patient's youth and the great amount of stenosis due to the inflammation, the fact that no anæsthetic was needed, and the size of the bone, very large for a boy's larynx.

Treatment of Coryza in Nurslings.—Acute coryza of nurslings is not by any means a harmless affection; the occlusion of the nasal passages and the accumulation of the secretion, by obstructing nasal breathing, have a number of sequelæ. Nutrition is seriously interfered with, and lung diseases are predisposed to. L. BALLIN (*Therapie der Gegenwart, 1905, Band vii., No. 2, p. 65*) thus describes his method of treating such cases: Adrenalin preparations are used in preference to cocaine, because of the slighter toxicity. Small cotton tampons are saturated with adrenalin solution, 1: 1000, and introduced into the nasal orifices alternately and kept there for two to three minutes; the mucous membranes become anæmic and the swelling decreases. This has to be repeated three to four times daily, as the adrenalin action persists only about four hours. A cure is usually produced in a few days. The fluid secretion diminishes quickly, and the crust formation must be treated with white vaselin. In weak infants, in whom the acute infection often passes into a persistent rhinitis, characterized by bloody mucopurulent

secretion, he employs, in addition to the adrenalin, ½ or 1 per cent. solution of silver nitrate, permitting 1 drop to enter each nasal orifice daily. The treatment must be continued beyond the time necessary to produce a cure. Of 75 children with coryza, 48 were treated according to this description; 6 developed bronchitis, 1 very weak infant died of bronchopneumonia; of the other 27, 12 developed bronchitis, 1 died of pneumonia. The nutrition of the latter group suffered much more than that of the former. Syphilitic coryza was also favorably influenced, and there have been no more deaths from lung complications in this disease since this treatment has been applied.

Hysterical Right-sided Deafness with Simultaneous Hyperæsthesia of the External Ear.—R. Doelger (*Münchener medizinische Wochenschrift*, 1905, Band lii., No. 14, p. 652) *reports the case of a* girl, aged twelve years, who, at the age of six, fell into a deep ditch; at eight she had pneumonia, followed by suppuration of the right middle ear, which lasted almost six months. For some months she complained of pain over and in the right ear, right-sided headache, dizziness, and nausea early in the morning. Examination of the ears showed the left one to be perfectly normal, the right mastoid tender, but no changes in the soft parts. The external auditory meatus was found extremely tender. The whispering voice could not be heard at all. Otherwise the examination was perfectly negative; the child was loquacious and bright, and there seemed no difficulty for her to continue a conversation. The case was believed to be one of hysteria, chiefly because of this normal general state and the tenderness of the lower bony part of the external meatus. Ice was applied locally and the mastoid rubbed daily with iodized vaselin. After *fourteen days of such treatment* she could hear perfectly well, only the hyperæsthesia remaining unchanged.

Cardiac Dilatation in Diphtheria.—H. Dietlen (*Münchener medizinische Wochenschrift*, 1905, Band lii., No. 15, p. 683), writing from Moritz's clinic in Greifswald, reports having examined 47 diphtheria patients by means of Moritz's orthodiagraph, the patients being in a reclining position, and having found 20 of these afflicted with myocardial conditions of various degree. In 15 of these (75 per cent. of the myocarditis patients and 32 per cent. of the total number) dilatations of varying degree were demonstrated with the orthodiagraph. This myocarditis appears usually in the first half of the second week, and in fully half the cases it leads to dilatation of some extent. In most of the cases dilatation begins with the myocarditis, but sometimes already before pulse changes are apparent; it increases gradually, reaching its acme with the height of the myocarditis, during the third week of the disease; it is associated usually with a diffuse apex beat, visible cardiac pulsation from the second to the seventh interspace, reduplication of the cardiac sounds, occasional murmurs, especially a blowing systolic sound and accentuation of the second pulmonic sound. Retrogression commences in the fourth week of the disease; in 2 instances out of 14 examined, it became complete within a short time, but in most it remained only partial. While complete recovery is possible, even in the worst cases, it is likely that in most of them it is only partial. The pulse changes are no criterion for the amount of the dilatation, some slight pulse changes being associated with marked dilatation and *vice*

versa. Percussion does not discover slight degrees of dilatation, and cannot be relied upon as the orthodiagraph can for absolute outlines. The prognosis is not absolutely bad, but the sight of such a heart calls attention to the danger in which such patients are constantly from paralysis of the heart. They should not be allowed to get up until the dilatation has receded to a point within the danger line.

OBSTETRICS.

UNDER THE CHARGE OF

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Studies in the Physiology of the Uterus.—FRANZ (*Zeitschrift fur Geburtshulfe und Gynakologie*, 1904, Band liii., Heft 3) gives the results of his studies in the physiology of the uterus in animals as follows: He found that the uterus is at all times strongly susceptible to the different kinds of electric currents. The reflex excitability of the pregnant uterus is much greater than that of the non-pregnant. When the uterus of a rabbit is excised it is not readily stimulated by the electric current. On the contrary, thermic influence strongly stimulates the excised uterus. If immersed in a bath of salt solution at the temperature of the body, the uterus first relaxes slightly and then vigorously contracts. If the temperature of the bath be raised uterine contractions increase, followed by dilatation. If the temperature is lowered, contractions increase both in vigor and in duration. Both electric and thermic stimuli often produce contractions, followed by relaxation in rhythmical form. There is no essential difference in the behavior of the human uterus and that of animals when excised from the body. In animals the vagina shows a similar reflex irritability to that of the uterus. Chloral hydrate given beneath the skin causes spontaneous contractions of the uterus in animals to cease; ergotine stimulates contractions and increases the tendency to rhythmical contraction. Atropine, whether given subcutaneously or into the veins, has little or no influence upon the uterus of animals. The reflex excitability seems somewhat diminished. Nicotine produces in the uterus in animals strong and continued contraction.

The Ability to Nurse the Child.—MARTIN (*Archiv fur Gynakologie*, 1905, Band lxxiv., Heft 3) gives the results of observations at the clinic at Stuttgart regarding the ability of women to nurse their children. Four-fifths of the patients under observation were blondes, one-fifth of them brunettes; 48 per cent. were primiparæ; 52 per cent. multiparæ. In 11.3 per cent. the breasts were only moderately developed; in 1.9 per cent. they were poorly developed, and in 2.1 per cent.

the nipples were flattened. In 1178 mothers 88.2 per cent. nursed their children; 10.5 per cent. nursed them partly; only 1.3 per cent. could not nurse them. As regards the results of nursing exclusively, 40.9 per cent. of the children had reached their original weight on the eighth day after birth, and 59.8 per cent. on the thirteenth day. When the children were partly fed and partly nursed 25.9 per cent. had reached or exceeded their original weight by the eighth day, and 35.6 per cent. by the thirteenth. The loss in weight for the first eight days after birth of children exclusively nursed was 18.6 grams to each 1000 of the body weight, and among those children partly nursed and partly fed the initial loss was 41.6 grams to 1000. By the thirteenth day children fed exclusively from the breast had exceeded their initial weight by 9.6 grams. It was found that those fed and nursed at the same time gained more slowly than those entirely nursed.

Infants fed exclusively by artificial means lost more rapidly after birth, and had a much smaller percentage of gain afterward. In the cases of those mothers who were greatly weakened by labor, as through hemorrhage or exhaustion, it was found that their children when they were able to nurse them gained more rapidly than did children partially nursed or children fed exclusively by artificial means. It was found that wounds of the nipples occurred in nursing mothers in 39 per cent. These were usually treated with success by strict asepsis and the use of some form of nipple shield. In 13 per cent. of the mothers there was painful swelling of the entire breast or some portion of it. This readily subsided with the use of a bandage and the lessening of liquids in the food taken. In 0.32 per cent. breast abscess occurred.

Nursing was interrupted or abandoned only in the severest diseases of the mother. Widespread tuberculosis, profound derangement of the blood, and those diseases which brought the child in danger were considered adequate indications for weaning. Syphilis of the mother was not considered a justifiable cause for removing the child from the breast.

In the last ten years it was found in the clinic that the number of women able to nurse a child had increased from 40 per cent. to practically 100 per cent. The ability to secrete milk for the child did not depend upon the size of the breasts, but upon the vigor of the reflex stimulation applied to the nipple. This might be either central or peripheral, the child furnishing the central reflex irritation through nursing.

Martin also calls attention to the psychic influences which affect lactation, notably the influence of depressing emotion in suspending the secretion of milk.

He believes it to be of especial importance that the child be placed to the breast as soon after birth as possible.

The Permanent Results of Delivery with the Use of Bossi's Dilator.—LICHTENSTEIN (*Archiv für Gynäkologie*, 1905, Band lxxv. Heft 1) has ascertained the condition of 18 patients in whose delivery Bossi's dilator was employed at Leopold's clinic at Dresden. In 11 of these cases no laceration of the cervix was found. In 4 there was slight laceration of the vaginal portion, in 2 extensive laceration of the vaginal portion, and in 1 bilateral laceration of the cervix. In none of these cases was there evidence of perimetritis following a tear. There was 1 case

of perimetritis upon the posterior aspect of the uterus with adhesions, but this could not be ascribed to the use of the dilator, as no laceration of the cervix had occurred. There had been, however, a chronic retroversion which had not been treated. In 1 case there was retroflexion of the uterus with descent. This condition gave rise to so much irritation that epileptic attacks apparently resulted. In 4 other cases there was retroflexion without other complications. In 1 case abortion occurred in a pregnancy following, and the patient had, after the use of the dilator, a small laceration in the vaginal cervix. There is, however, no evidence that abortion resulted from this. As the patient had severe eclampsia, it is more probable that her abortion was caused by some lesion of the kidneys Five of these patients had subsequently been delivered in their own nomes, and 4 were found on investigation to be in a pregnant condition. Of the 5, 3 had no lacerations, 1 had a small tear of the cervix, another had an extensive tear. Of the 4 patients found in a pregnant condition, 2 were in the seventh, 1 in the eighth, and 1 in the second month. One of these had contracted pelvis. Of the 17 women, 10 had subsequently become pregnant and 1 of these only had aborted. In the remaining 7 there were 4 cases of retroflexion, and 2 of these were complicated by prolapse of the uterus. As regards the symptoms complained of by these patients, 10 were without pain or distress, 3 had vaginal catarrh and constipation, 1 had disability resulting from an operation upon the ovary, 1 had metrorrhagia, but had no laceration of the cervix, and 1 was intensely nervous, for which no cause in the pelvic organs could be found. As regards ectropion, it was present in 3 cases only and to a mild degree. The result of the investigation of these patients showed that the use of Bossi's dilator in skilful hands is not followed by permanent damage to the patient's health.

An Abnormally Long Umbilical Cord.—GOTTSCHALK (*Zentralblatt fur Gynakologie*, 1905, No. 5) describes a placenta with an umbilical cord 72 cm. long. The child was born in breech presentation living, the mother having a normal pelvis. The cord was filled with blood and twisted into many spirals. It was wrapped about the child, and especially about the neck, in such a manner as to make breech presentation the only way in which the child could be born living. Compression of the vessels of the cord produced the transudation of serous fluid. The entrance of the breech in the pelvis and extraction of the head were rendered more difficult by the coiling of the cord.

Ligature of the Spermatic and Hypogastric Veins in Puerperal Pyæmia.—BUMM (*Zentralblatt fur Gynakologie*, 1905, No. 5) has ligated the spermatic and hypogastric veins in 2 cases of patients suffering from puerperal pyæmia. In the first the patient had frequent and violent rigors, the veins were traced upward into the abdominal cavity, and were ligated very carefully to avoid opening the vessels. In the second case chills ceased entirely after the operation.

The Ultimate Disposition of Embolic Placental Cells.—SCHMORL (*Zentralblatt fur Gynäkologie*, 1905, No. 5) has examined the bodies of 150 women dying in various stages of pregnancy and in the puerperal state. He paid special attention to the lungs in these bodies. He found that placental cells were carried into the lungs as emboli very frequently.

In 83 cases of eclampsia these cells seemed to be unusually abundant. In women dying in early pregnancy placental cells were found in the lungs much less frequently than in those perishing at full term. Microscopically the lungs were unchanged.

In cases of vesicular mole these cells are also found, and in process of rapid growth.

Shall the Practising Physician Be Advised to Employ Bossi's Method of Delivery?—In the *Archiv fur Gynäkologie*, 1905, Band lxxv., Heft 2, DÜHRSSEN strongly urges against rapid dilatation of the unaltered cervix by Bossi's dilator. He very naturally contrasts with this his method of delivery by vaginal Cæsarean section. He reports several interesting cases.

The first case was that of a primipara with a flat pelvis, and who had gone considerably over the usual time for delivery; had been long in labor and had a greatly distended lower uterine segment; the head had failed to descend, but was wedged in the pelvis. To relieve the tension in the lower uterine segment and to enable delivery to take place, Dührssen performed vaginal Cæsarean section. He first made a free incision upon the right side of the vulva and tied bleeding vessels, then the cervix was seized by forceps, the inferior portion divided, and also the lower posterior walls of the vagina, the anterior lip was then divided, and also a portion of the anterior vaginal wall 5 cm. in length. The bladder was then loosened from the vagina, when the hand was readily introduced into the uterus, and version readily performed. A large child was delivered, asphyxiated, but was revived. The placenta was delivered by hand; the uterus packed with gauze; incisions and lacerations closed with catgut. The operation consumed forty minutes. Mother and child made a good recovery.

This case seems to us one in which the operator took a very great and unjustifiable risk. A contracted pelvis and an overgrown child cannot usually be overcome by incision into the soft parts; the indication was rather for abdominal Cæsarean section than for vaginal, and the operator was more fortunate than wise in his procedure. In his accustomed hands this attempt was made successfully, but an operator less experienced with this method might have torn the uterus to a fatal extent during delivery.

Dührssen also reports 2 cases of threatened eclampsia; 1 in which labor was brought about by the insertion of a bag; while the second patient came into spontaneous labor after the introduction of a bougie. His purpose in reporting this case seems to be to show that he does not in all cases perform vaginal Cæsarean section, and that there are also cases which can be delivered before the reports of his cases.

His fourth case was one of eclampsia at seven months in a multipara, in whom the cervix had been lacerated and was enlarged and very hard; the cervical canal admitted one finger. In this case vaginal Cæsarean section followed by version was performed. The patient made a good recovery.

Dührssen states that after dilatation with Bossi's dilator, 37.5 per cent. of the children are lost, and 22.6 per cent. of mothers. In 137 cases of vaginal Cæsarean section he has lost 19, a mortality of 14 per cent.

It must be remembered that the results of vaginal Cæsarean section do not compare favorably with abdominal Cæsarean section. In cases

of normal pelvis where the fœtus is not abnormal in size in the hands of an operator accustomed to perform vaginal section, this method may be useful. It has, however, its very decided limitations, and should not, we believe, be applied in cases of considerable pelvic contraction.

GYNECOLOGY.

UNDER THE CHARGE OF

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ASSISTED BY

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Results of Alexander's Operation.—FUCHS (*Zentralblatt fur Gynakologie*, 1905, No. 20) reports 220 cases from Werth's clinic. In 69 patients who were kept under observation a recurrence took place in 14.4 per cent. No case of hernia was noted. The canal was laid open and Bassini's method was followed in two-thirds of the cases. There were 7 cases of temporary displacement following labor, while in 6 other women the uterus remained in its normal position.

Division of the ileoinguinal nerve was seldom followed by subsequent pain.

The writer is opposed to shortening of the round ligaments after the separation of adhesions Vaginal fixation is preferable in cases of prolapsus with retroflexion.

Desmoid Tumors of the Abdominal Wall.—PFEIFFER (*Beitrage zur klin. Chirurgie*, Band xliv., Heft 2) reports 40 cases from Bruns' clinic and 360 from the literature; 89.4 per cent. occurred in women, the majority having borne children. The umbilicus was never affected. The microscopic diagnosis between fibroma and fibrosarcoma is difficult, nor does the occurrence of round and spindle cells in the growth necessarily imply malignancy. The writer has never observed a case of pure myoma, though he has rarely met with fibromyxoma and fibromyoma, and one case each of pure myxoma and myxosarcoma.

He explains the etiology of desmoids, like Ribbert, by the theory that after hypertrophy of the abdominal muscles and connective tissues following labor the walls become overstretched, herniæ and spaces occur, which, as the result of hyperæmia and cell-proliferation, are filled in with neoplasms. Trauma is doubtless a common etiological factor. It is not necessary, however, to infer with some authors that these tumors have their origin in hæmatomata.

Desmoids cause but little pain, which is sometimes most marked at the time of menstruation. Rapid growth is not an indication of malignancy. They may undergo degeneration and even suppurate and discharge through the skin.

An important diagnostic point is the sharply circumscribed character of the neoplasm, and its immobility when the abdominal muscles are contracted. They are distinguished from intraperitoneal tumors by the fact that the greater portion of the mass is external and that they do not change their position with change of posture and inspiration. Abscesses, cysts and gummata must be considered.

The writer advises removal of the tumors, even in the early months of pregnancy, on account of their tendency to grow rapidly in this condition. In the later months interference would be justifiable only if the neoplasm was so large as to threaten to interrupt the pregnancy, or hemorrhage or suppuration occurred. In excising it the surgeon should extend his incisions into healthy tissue, whether the peritoneal cavity is opened or not. Gaps in the peritoneum can be filled in with flaps from the neighboring serous membrane or omentum, while plastic operations will supply defects in the muscular wall.

The primary mortality is only 4.55 per cent.; the prognosis being most favorable in cases in which the peritoneal cavity is not opened.

In 107 patients who were kept under observation, local recurrence occurred in 21.2 per cent. in women and 50 per cent. in men, the more unfavorable statistics in the latter being due to the fact that the growths were nearly always sarcomatous. Repeated operations have been performed with an ultimate good result, since many of the recurrent neoplasms are pure fibromata.

Enterostomy in the Treatment of Peritonitis.—GEBHARD (*Deutsche Zeitschrift für Chirurgie*, Band lxxiv., Heft 1 and 2) believes that the principal danger in septic peritonitis is intestinal infection, the retained intestinal contents constituting septic foci, from which the peritoneum is poisoned. He accordingly advises enterostomy, in order to drain away this infectious fluid.

He reports 8 cases of incipient peritonitis, in which he adopted this method of treatment, in all of which the establishment of an intestinal fistula was followed by arrest of the general infection and subsequent cure.

Spontaneous Replacement of the Retroverted Uterus.—KLEINWACHTER (*Zeitschrift für Geb. u. Gyn.*, Band liv., Heft 1), observing 366 cases of uncomplicated movable retrodisplacement for a period of years, found that the uterus returned to its normal position spontaneously in 11.2 per cent., and remained thus. He believes that this occurs more frequently than is usually supposed.

Cancer following Removal of Benign Ovarian Tumors.—SCHRŒDER (*Zeitschrift für Geb. u. Gyn.*, Band liv., Heft 1) adds the following case to the 6 already reported: Three and one-half years after the removal of a small multilocular cystadenoma of the left ovary (the right being also extirpated, as it was cystic) the abdomen was reopened on account of ascites. At the same time a portion of the thickened abdominal wall at one side of the old cicatrix was excised, and when examined microscopically proved to be adenocarcinoma. The peritoneum was studded with cancerous nodules, hence the inference that infection had occurred during the removal of the pseudo-mucin cyst, with subsequent malignant degeneration.

Pneumococcus Infection of a Dermoid Cyst.—RISSMANN (*Deutsche med. Wochenschrift*, 1905, No. 13) reports the case of a woman, aged thirty-two years, who, on the eighth day after the inception of lobar pneumonia continued to have a high pulse-rate and temperature. She had had an abdominal tumor for several years, which was removed and proved to be an immense dermoid cyst. Pure cultures of pneumococci were made from the cyst contents. Since there were no intestinal adhesions, the writers believe that the infection took place either through the blood, or secondary to the pneumococcus peritonitis.

Lutein Cells in Pregnancy.—SEITZ (*Zentralblatt fur Gynäkologie*, 1905, No. 19), from an examination of the ovaries of 17 women who died during or soon after pregnancy, concludes that the atresia of the follicles differs from that which occurs in the non-pregnant in the marked development of the cells of the theca internum, usually known as lutein cells. As long as uterine pregnancy persists (even when pathological) ovulation ceases and the follicles degenerate. This applies to chorion epithelioma, as opposed to cancer of the uterus. The writer ventures to express no theory as to the physiological significance of this increase of lutein cells during pregnancy.

Foreign Bodies in the Uterus.—KNOOP (*Zentralblatt fur Gynakologie*, 1905, No. 19) reports 2 cases and cites nearly 100 others from the literature.

In the first case a hairpin was removed from the uterus, having been introduced six years before. The patient was fifty-seven years old; had ceased to menstruate ten years before, and was sent to the clinic for supposed cancer of the corpus uteri, on account of an offensive discharge.

Sterilization by Resection of the Tubes.—REIFFERSCHEID (*Zentralblatt fur Gynäkologie*, 1905, No. 19), in referring to 2 cases of Kustner's, in which women became pregnant after portions of the tubes were ligated and excised, describes the method practised unsuccessfully at the Bonn clinic. At the time of a second Caesarean section each tube was tied with silk in two places near the uterine end and an inch was excised, after which the uterine stumps were turned in and sutured, but the patient, nevertheless, became pregnant again and premature labor was induced. The writer accordingly agrees with Kustner that the only sure way to prevent future impregnation is to excise the tubes entirely, as in pyosalpinx.

The indications for the operation must be strictly defined, and it should be performed only with the consent of the patient's husband.

Action of Streptococcus Pyogenes on the Vaginal and Uterine Mucosa.—HEINRICIUS (*Archiv fur Gynakologie*, Band lxxiv., Heft 22), from experiments on rabbits, concludes that when the superficial epithelium is intact the bacteria are unable to make their way through or between the cells into the subjacent muscular tissue, the reverse being true if the epithelium becomes desquamated or is exfoliated. The writer also finds that the lymphatics are the usual channels of peritoneal infection in cases of septic endometritis. The factor of individual susceptibility to infection was clearly shown in experiments upon rabbits, which is

often borne out by clinical observations in the human subject, in whom general sepsis may develop without discernible local lesions.

Cauterization in Inoperable Cancer of the Uterus.—LOMER (*Zentralblatt für Gynäkologie*, 1905, No. 19) reported to the Hamburg Obstetrical Society a case of cauterization of an inoperable cancer in which the uterus, previously firmly adherent, became so movable that total abdominal extirpation was easily performed a few days later, no involvement of the pelvic glands and tissues being found. The speaker referred to a similar case in which the uterus was successfully extirpated five years after curettement and cauterization.

In the discussion which followed opinions differed, both as to the value of cauterization and the frequency of metastasis in the lymph nodes.

Clinical Observations in Ovarian Tumors.—LIPPERT (*Archiv. für Gynäkologie*, Band lxxiv., Heft 2) analyzes 638 cases occurring in the Leipzig clinic in the course of seventeen years, the operative mortality being 5 per cent., or 3.7 per cent. in benign and 13.13 per cent. in malignant growths.

The writer agrees with Byford that ovarian tumors are more frequent in married women—in fact, over 96 per cent. of the papillary variety occurred in those subjects. Menstruation was comparatively little affected, even with double cysts. Amenorrhœa was occasionally noted. Adhesions were present in 48.5 per cent.

Abdominal ovariotomy was performed in 81 per cent., and explorative incision alone was performed in only 3.9 per cent. of the benign growths and 23.5 per cent. of the malignant.

As to the question of removing the opposite ovary, its cystic condition and the age of the patient were the principal points considered. A second ovariotomy was performed in 3.3 per cent.

Etiology of Purulent Tubo-ovarian Cysts.—SCHAFFER-HEIDELBERG (*Archiv für Gynäkologie*, Band lxxiv., Heft 2) refers these cysts to a former inflammatory process. He believes that the fimbriated end of an inflamed tube becomes adherent to the ovary, and that suppuration occurs in the latter, so that it is transformed into an abscess. Interuterine infection as a result of direct applications or the use of tents may cause the lighting up of old tubal inflammation. Vaginal incision and drainage is the treatment recommended.

Early Diagnosis of Tuberculosis of the Uterus.—SCHATTLANDER (*Monatsschrift für Geb. u. Gyn.*, Band xxi., Heft 1 and 2) does not believe that polymorphism and multiplication of the cells of the glands and endometrium is sufficient to justify the suspicion of commencing tuberculosis in the absence of bacilli, unless gonorrhœa or recent abortion can be positively excluded. Moreover, the demonstration of the presence of tubercle bacilli, although an important aid to diagnosis, is not confirmatory as in the finding of giant cells.

Innervation of the Female Pelvic Organs.—JUNG (*Monatsschrift für Geb. u. Gyn.*, Band xxi., Heft 1 and 2), from a careful study of serial sections of fœtuses, arrives at the following conclusions: In the new-

born a large ganglion is found in the parametric tissue opposite to the junction of the cervix and vaginal vault, from which chains of ganglia radiate downward to the pelvic floor and upward to the ureter and vesicouterine pouch. These communicate with the ganglia behind the rectum, around the ovary, and with the renal ganglia.

This cervical ganglion does not form a plexus, but constitutes a mass of plexuses to which all the genital nerves converge. It is not as large as Frankenhäuser pictures it, but is composed largely of connective tissue. The entire vagina is surrounded by a network of nerve fibres and ganglia, situated partly within the paravaginal connective tissue, which communicate with the large ganglion before mentioned.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

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Diphtheria Antitoxin in the Treatment of Goitre.—Dr. Robert T. Legge, of McCloud, Cal. (*Journal of the American Medical Association*, April 22, 1905), reports a case of a lady in whom he had injected 1500 units of antitoxin on two successive days in the treatment of a moderately severe pharyngeal diphtheria, contracted in nursing a fatal case of the disease in a child. This lady had exophthalmic goitre of eight years' duration, with all its classic symptoms. Four months later Dr. Legge noticed that the large tumor of the neck had disappeared and that the eye symptoms were a trifle better. After a lapse of one year there had been no return of the enlargement. Two cases are reported in which the antitoxin was given purposely, and in which marked reduction of the tumors took place in from three to five months after two injections at about ten days' interval.

Stenosis of the Nasal Cavity Caused by Contraction of the Palate and Abnormal Position of the Teeth; Expansion by the Treatment of the Maxilla.—At the Fourth International Dental Congress held at St. Louis, Mo. (*Dental Cosmos*, May, 1905), Dr. G. B. Maercklein, of Milwaukee, read a paper upon this subject and recommended an apparatus for the purpose suggested by Dr. Schroeder by which he had performed a great number of expansions of the maxilla in treating cases of prognathism, and always succeeded in effecting nose breathing where it had been impossible before.

Artificial Noses.—The *Dental Cosmos* for May, 1905, contains interesting illustrated articles by Dr. Louis Ottofy, of Manila, P. I., who made an artificial nose for a Chinaman; Dr. W. H. Baird, of Burlington, Iowa, who supplied an artificial nose to a lady; and Dr. Lee Baker, of

Port Townsend, Wash., who supplied a nose to a girl, aged sixteen years, who had lost palate, as well as nose, and in whom the artificial contrivance not only improved the personal appearance, but enabled her to talk and sing. The patient last mentioned had continually complained of cold air keeping the soft tissues so inflamed that she suffered a great deal of pain, but since the artificial appliance has been in use the tissues have resumed their normal condition and she has increased greatly in weight and general health.

Sarcoma of the Nose.—Dr. William H. Ballenger (*Laryngoscope*, February, 1905) reports a case of a young lady with a sarcoma of the nose in which he first removed nearly the whole of the right inferior turbinate and submitted it to a pathologist for examination, receiving the report that the process was tuberculous. This report being unsatisfactory to him, he removed some more tissue and received a report that it was granulation tissue. Feeling that the case was one of sarcoma, despite the findings of the pathologist, he decided to operate by the radical method of sawing through the maxillary bone and turning the nose down over the mouth. In this way he was able to remove a large amount of necrotic and diseased tissue. After submitting specimens to the pathologist, he received a report that the disease was sarcoma. There had been no recurrence at the date of the report.

Cleft Palate.—At the International Dental Congress, held at St. Louis, Mo., Dr. T. W. Brophy, of Chicago (*Dental Cosmos*, May, 1905), exhibited some patients and read a paper on "The Radical Treatment of Congenital Cleft Palate." In cases associated with harelip, he advocated operating upon the palate first, in order to have more room for this procedure. He operates at as early a date as practicable, even in children but ten days old, and would always operate, if possible, before the third month of age.

At a meeting of the Laryngo-Rhino-Otological Society of St. Petersburg, (*Archives internationales de laryngologie, d'otologie et de rhinologie*, Mai-Juin, 1905) Prof. Simanovski presented a patient in whom he had successfully utilized the mucous membrane and periosteum of the septum narium in forming a flap in a plastic operation of a portion of the fissure remaining uncovered after previous operations.

Removal of Adenoids.—At a late meeting of the American Laryngological, Rhinological and Otological Society, held in Chicago, Dr. George L. Richards (*Laryngoscope*, February, 1905) showed a very large adenoid removed from a child aged six years, which had given rise to asphyxia on spasmodic closure of the jaw just as the child was apparently under complete ether anæsthesia. He had to resort to artificial respiration, hypodermic injections, forcible opening of the jaw, and traction of the tongue, in order to resuscitate his patient.

Primitive Tuberculosis of the Turbinate Body.—At a recent meeting of the Laryngological Association of Vienna, Dr. Glas (*Archives internationales de laryngologie, d'otologie et de rhinologie*, Mai-Juin, 1905) presented microscopic preparations made from the anterior portion of a middle turbinated bone amputated for the purpose of gaining access

to the opening of the left maxillary sinus, which had discharged pus for two months, in a young lady, aged twenty years. Although the slightly hypertrophied mass did not show ulceration nor granulations, microscopic examination revealed portions containing lymphoid tubercles and epithelial cells with partial caseation and a large number of giant cells. The secretion from the sinus, however, did not furnish any bacilli. The lungs were apparently normal.

Disease of the Maxillary Antrum of Dental Origin.—At a late meeting of the American Laryngological, Rhinological and Otological Society, held in Chicago, DR. GEORGE L. RICHARDS (*Laryngoscope*, February, 1905) presented a specimen of a capped tooth which had set up antral disease. He had seen it stated in some publications that dentists had been held responsible for setting up antral trouble by capping teeth which ought to have been treated otherwise.

Tuberculosis of the Pharynx and Rhinopharynx.—DR. GLAS (*Archives internationales de laryngologie, d'otologie et de rhinologie*, Mai-Juin, 1905) reported a case of tuberculosis of the pharynx and rhinopharynx in a man, aged twenty-nine years, who, the previous Easter, had had a bone incarcerated in his pharynx for some eight days. When examined, the apex of the left lung was in a suspicious condition, and the left vocal band presented a slight thickening near the anterior commissure. The mucous membrane of the pharynx was covered with several masses of reddish granulations with grayish nodules upon them here and there. Behind the right palatopharyngeal fold was a cicatrix, 2 cm. in length, corresponding with the original wound. There was ulceration at the orifice of the left Eustachian tube and another at the vault of the pharynx. Scrapings from some of these structures revealed the presence of tubercle with necrosed centre containing large giant cells and some bacilli, but neither the mucopurulent secretion nor the scabs contained bacilli.

Laryngeal Tuberculosis.—DR. E. S. BULLOCK, of Silver City, N. M. (*Monthly Cyclopædia of Practical Medicine*, March, 1905) extols mopping the tissues twice a day or oftener, with a weak solution of formaldehyde, 2 to 10 drops of the 4 per cent. solution to the ounce of water; or as the second best application, with trichloride of iodine, ½ to 2 grains to the ounce of distilled water.

Unusual Foreign Body in the Larynx.—DR. ZIANOURY, of Constantinople (*Archives internationales de laryngologie, d'otologie et de rhinologie*, Mai-Juin, 1905), reports an instance of the inspiration of pieces of eggshell in the larynx of a boy, aged six years, while taking soup. The foreign body, supposed to be a fragment of bone, evaded detection by palpation, laryngoscopy, and radioscopy, both before and after tracheotomy. A thyrotomy was performed to expose the interior of the larynx, when some pieces of eggshell were caught upon the gauze swab used for mopping the blood. It is supposed that the thinness of the foreign body prevented its recognition under the *x*-rays.

Syphilitic Tracheal Stenosis in a Six-year-old Child.—DR. LEE MAIDMENT HURD, of New York (*Laryngoscope*, February, 1905), reports a case of tracheal stenosis in a boy, aged six years, who had been infected

by a kiss at three years of age. Low tracheotomy revealed a syphilitic infiltration below the larynx, with an aperture about as large as an ordinary silver probe. Alternate use of the tracheotomy cannula and of a long intubating tube, with protracted use of mercurial inunctions and iodide of potassium, successfully relieved the case from all manifestations of syphilis at the end of seven months' treatment. This child took on an average of 180 grains of potassium iodide a day for more than a month and was still taking 180 grains in the day at intervals at the date of the report.

Syphilitic Ulceration of the Œsophagus in an Infant Eighteen Months of Age.—Dr. Fackeldey (*Annales des maladies de l'Oreille du larynx, du nez et du pharynx*, Mars, 1905) has reported the case of an infant with dysphagia attributed at first to foreign body in the œsophagus. Œsophagoscopy revealed an ulceration undergoing cicatrization. Recovery followed antisyphilitic treatment and dilatation of the cicatrix.

Foreign Bodies in the Œsophagus.—Dr. Carl E. Black, of Jacksonville, Ill. (*American Medicine*, January 28, 1905), reports 2 cases of foreign bodies in the œsophagus in children, one a pin and the other a half-inch iron washer, the location of both of which were determined by fluoroscopy and skiagraphy. Attempts at extraction were made under the guidance of the fluoroscope successfully as regards the pin; unsuccessfully as regards the washer.

In the manipulation the head of the patient was drawn over an end of a table and held in position by an assistant. The Roentgen-ray tube was placed beneath the patient, so that the operator could hold the fluoroscope with his left hand and direct the forceps with the right hand.

DERMATOLOGY.

UNDER THE CHARGE OF

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Lichen Spinulosus.—H. G. Adamson (*British Journal of Dermatology*, March, 1905), under this name, describes clearly an affection of the skin occurring in children, usually boys, characterized by formation of fine filiform spines arranged in groups distributed over the trunk and limbs. The filiform spines arise from the pilosebaceous follicles, the mouths of which are slightly raised to form pinhead-sized papules, either of the normal color of the skin or slightly red. They are unaccompanied by itching or other subjective symptoms, and there is little or no disturbance of the general health. Histologically, the lesions show the

process to be essentially a hyperkeratosis of the follicle, perifollicular inflammation being absent or but very slightly existent. The difference between this disease and lichen pilaris is carefully pointed out. It is well known in England, but is not so clearly described in France. Cases are occasionally met with in this country.

Anatomy of Dermatitis Papillaris Capillitii (Kaposi).—JOSEF GUSZMAN (*Dermatologische Zeitschrift*, March, 1905) writes a lengthy article on the subject, concluding by stating that this disease consists of a chronic granulation process, which in the beginning has its seat in the upper layers of the skin, gradually extending downward through the involvement of circumscribed perivascular areas. The sebaceous and sweat glands in the beginning show no changes, but are later involved secondarily, with the result finally of being, to a greater or less extent, destroyed and disappearing. The characteristic peculiarity of this disease is the existence of granulation tissue without tendency to disintegration, but taking on a remarkable consistence in the form of a fibrous mass, causing in the end a disappearance of the hair follicles and hairs.

Liquid Air and its Possibilities.—S. G. TRACY (*Medical Brief*, February, 1905) states that even when it became possible to make liquid air at a comparatively inexpensive price (it is now sold at ten dollars per gallon, as made by the Tripler system in New York), its usefulness would have been limited or of no avail in medicine, except for the invention of a bulb by Dewar, used to hold this extraordinary product. It is a glass vessel with two walls, the space between being exhausted to high vacuum. When liquid air is contained in one of these it evaporates at one-fifth the rate it does in an ordinary glass receptacle. When liquid air is applied to the skin and tissues of the body the parts are immediately frozen. At first there is anæmia of the part, followed by reddish reaction or slight inflammation. If the freezing has been severe, there results a bleb. Liquid air is not only a local anæsthetic, little or much anæsthesia being produced by it in a few seconds' time, and the anæsthesia lasts longer and is followed by less sloughing than any other freezing mixture. Slight pain attends the application. After anæsthesia has been obtained a minute or more should elapse, as the tissues may be too hard for operation. It is applied with a swab of absorbent cotton on a wooden applicator, firm pressure being used, the application lasting from twenty to sixty seconds, depending on the tissues to be affected. The histories of several cases of cutaneous carcinoma and nævus are related. In lupus erythematosis, where the lesion is not too large, the author thinks it more satisfactory than any other remedial agent.

Treatment of Lupus, Angioma, Nævi, and Keloidal Scars with Radium.—WERNER and HIRSCHEL (*Deutsche med. Wochenschrift, 1904, No. 42*) report good results with raying by means of radium daily or every other day for several hours over a period of from three to fourteen days. The remaining scars were white, smooth, and satisfactory, and recurrences were not observed in from six to nine months afterward.

Paget's Disease and its Relation to Carcinoma of the Breast.—H. C. JACOBAEUS (*Virchow's Archiv*, Band clxxviii., Heft 1) concludes from his studies that the so-called Paget's disease is a carcinoma, from the

beginning originating in the glandular epithelium of the ducts. The Paget cells are not carcinomatous, degenerated, localized, epidermis cells, but are glandular carcinomatous cells that have wandered. Their occurrence, isolated or in little groups, is the result of a peculiarity of the epidermis.

Treatment of Cutaneous Diseases by Combined Ointment and Phototherapy.—L. STEINER (*Journal of the American Medical Association*, June 11, 1905, p. 1970) states that far better results are obtained when ointments are used at the same time as reflected electric light than when either is used alone. Numerous experiments at Hoffmann's clinic at Leipzic are reported, nearly 40 cases being recorded. The active hyperæmia induced probably accounts for the good results, this being supplemented by the bactericidal action of the rays. When used with a sulphur or mercurial ointment, eczema, cycosis, acne, and other diseases were more rapidly and favorably influenced than by any other method of treatment known to the author.

Treatment of Scalp Ringworm.—T. C. FOX (*Practitioner*, April, 1905) recommends the employment of the old and well-known treatment by means of croton oil, advising the use of an ointment 1 drachm to the ounce. This is rubbed in daily or as often as necessary to excite the desired amount of inflammation. The inflammatory products, including crusts, are cleaned off as they form.

Hyperidrosis Axillæ Successfully Treated by X-rays.—G. H. STOVER (*Archives of Physiological Therapy*, April, 1905) relates the history of a remarkably severe instance of this affection occurring in a young married woman, the fluid poured out being enough to saturate the clothing to the waist and to drip. The quantity was estimated at two quarts daily. This condition had existed for many years, in spite of all manner of treatment, local and internal. A low tube, four inches from the skin, exposing each axilla on alternate days for ten minutes, was used. After the trunk exposure the skin showed slight pigmentation, and after the twenty-fourth exposure there existed slight erythema—never more than a mere blush. At the thirty-first exposure the perspiration was distinctly less; at the thirty-eighth there was very great improvement; at the forty-fifth the hair fell out rapidly. At no time did the hair become gray or white. Treatment was now intermitted, the perspiration being somewhat in excess of the normal amount. Four months later the axillary hair had returned. Twelve exposures were now given on succeeding days. After the eighth pigmentation appeared and the hair fell again, and there was slight erythema at the end of the series and the perspiration had stopped entirely.

Cutaneous Anthrax Successfully Treated by Sclavo's Serum.—LOCKWOOD and ANDREWS (*British Medical Journal*, January 7, 1905) report a case occurring on the cheek of a man who worked with horse hair; 40 c.c. of the serum were injected into the abdomen, no other remedies being employed. The temperature at the time was 102.4°, and the pulse 92, but during the night the temperature fell to 98.6°. During the following two days no further improvement was noted, but on the third day there was distinct improvement. No further dose of the serum

was given, and seventeen days later only a small granulating sore remained, thus proving the efficiency of the serum treatment.

Radium Treatment of Cancer.—A. EXNER, of Vienna (*Deutsche Zeitschrift fur Chirurgie* and *Journal of the American Medical Association*, April 15, 1905) describes a case of carcinoma of the cheek of a woman, aged seventy-three years, which had been declared inoperable by some surgeons. Radium was used daily for a month, then the lesion was curetted and radium again applied for twelve days. He grinds 60 mg. of radium bromide with about 1.5 c.c. of xylol and adds 2 drops of dammar. This mixture is placed in an aluminum box about 1.5 cm. square. The radium dust settles to the bottom very evenly when stirred. The strength of the radium used equals twelve Holzknecht units in four hours.

Diseases of the Skin Connected with Errors of Metabolism.—L. DUNCAN BULKLEY (*Medical Record*, November 26, 1904) gives an interesting article on a subject but little understood. He concludes from his studies, based on many thousands of cases, that metabolism is principally affected by the kind of nutriment taken, the action of the digestive organs of the nervous system. Certain cutaneous lesions or eruptions have been credibly reported as connected with or dependent upon the generally recognized metabolic conditions of gout, rheumatoid arthritis, diabetes, obesity, and scrofulosis; but as yet no absolute statements can be made as to the necessary connection of the two, for the same eruptions occur in several of the metabolic affections. The idiosyncrasy of the individual and many causative elements, external or internal, nervous, etc., often determine which form of skin disturbance or alteration shall take place. Errors of diet, disorders of digestion, faulty excretion, and nervous derangement often find their ultimate expression or mode of action through the faulty metabolism induced thereby. Metabolic errors are manifested not only through the lungs, intestines, and kidneys, but also through the skin. The author finally calls attention to the importance of complete and minute urinary analysis in discerning metabolic errors and in establishing proper therapeutic measures for the cure of many diseases of the skin.

Treatment of Alopecia Areata.—KROMAYER (*Deutsche med. Wochenschrift*, 1904, No. 31) reports the cure of 6 rebellious cases of this disease by means of cold iron light (Triplet or dermo lamp), the rationale of the treatment being found in the artificial inflammation of the skin. The latter is provoked by frequent use of the lamp up to a point of considerable inflammation of the skin, when a rest is allowed, and then the same treatment begun again.

Effects of Roentgen Ray on Cancer.—R. H. VOSE and W. C. HOWE (*Journal of Medical Research*, January, 1905) made observations on 120 cases. The effect on ulcerated surfaces was in the beginning that of stimulation as evidenced by the granulations, but later the epithelium is destroyed and granulation retarded. Cutaneous carcinoma treated by the *x*-ray shows a degeneration of the tissues affected, which, however, is not to be distinguished under the microscope from degenerations of the skin due to other causes. As to the bloodvessels, there occurs an

endarteritis; where healing occurs there is new formation of blood-vessels, as takes place elsewhere during the process of repair, and there is an augmentation of the elastic tissue. The authors arrive at the conclusion that the cure of carcinoma by *x*-ray is due to destruction and exfoliation, and that this fact limits the value of the agent to superficial cases.

The Finsen Method.—MILTON FRANKLIN (*Medical News*, December 31, 1904) discusses the subject of the treatment of the tuberculous diseases of the skin by means of the ultraviolet or chemical rays of the spectrum, and describes the apparatus used. The latter should be free from glass and powerful, if good results are to be expected. The use of the *x*-ray in lupus and kindred diseases, he thinks, has been too limited to regard this agent as of great value, whereas, the Finsen apparatus has positively cured these diseases in a large number of instances, and he consequently looks upon it as preferable to the *x*-ray method.

Quinine-Iodine Treatment of Erythematous Lupus.—M. OPPENHEIM (*Wiener klin. Wochenschrift* and *Journal of the American Medical Association*, March 25, 1905) speaks in praise of Holländer's method of treating this disease by quinine internally and iodine externally, the patients being 6 cases in Finger's clinic, 2 being cured and the others much benefited. Experiments made upon animals led the author to the conclusion that impregnation of the body with quinine attracted the iodine employed locally to the cutaneous lesions, where it acted specifically. Bisulphate of quinine, in increasing doses up to 1 drachm daily, was used, the local lesions being brushed with tincture of iodine twice daily after removal of the crusts with alcohol and ether, with occasional interruptions. Treatment was continued for a month.

Radiotherapy of Carcinoma.—DJEMIL PASHA (*Revue de Chirurgie*, from *Journal of the American Medical Association*, March 11, 1905) reports 7 cases of carcinoma treated with *x*-rays with good results; 3 were cured, 3 were benefited, and 1 not relieved. He advises this method as a palliative treatment in advanced deep-seated carcinoma, its influence on the cachexia being very favorable.

Treatment of Lupus by X-rays and the Finsen Lamp.—F. H. JACOB (*Medical Electrology and Radiology*, November, 1904) arrives at the conclusion that better results are obtained by the use of accessory treatment with the rays and the light, especially the topical use of carbolic acid just prior to using the light; also pyrogallol in the form of ointment or plaster, 20 grains to the ounce. Drugs of this kind will remove the larger lesions of lupus, but the minute outlying lesions so difficult to reach are best healed with the light. The author regards the scar following the Finsen light as better than that of the *x*-rays. The chief disadvantage of the *x*-ray is that it is prone to produce enlargement of the bloodvessels with structural changes, in the form of telangiectasis. This unfortunate result may be obviated by employing the rays for short periods at each sitting or for a long time at long intervals. While the beauty of the scar following the Finsen light is esteemed, the slowness and tediousness of this treatment is emphasized.

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Changes in the Circulatory Apparatus, Especially in the Peripheral Vessels in Typhoid Fever.—WIESSEL (*Zeit. f. Heilk.*, 1905, Band xxvi. p. 107) has made a study of the histological changes in the heart and peripheral vessels in cases of typhoid fever. All those cases were excluded in which the vessels showed chronic endarteritis or in which some complication might mask alterations due to the typhoid fever alone. Besides the heart muscle, the aorta, pulmonary artery, radial, iliac, and femoral arteries were particularly investigated. The heart muscle showed fatty and parenchymatous degenerations with certain changes in the cell nuclei; but no new facts of special moment were brought out during this part of the investigation. Very important alterations were, however, found in the walls of the peripheral vessels. This subject has attracted the attention of certain French writers who describe lesions of the intima, especially in the coronary arteries. The main changes noted by the author were not in the intima, but in the middle coat of the vessels. Both the muscle fibres and the elastic tissue proved to be diseased. The muscle fibres showed atrophic changes, together with an increase in the intercellular connective tissue with proliferation of these cells. But by far the most pronounced changes were noted in the elastic tissue. The elastic fibres were broken, torn, and frequently stained poorly. In places the degenerative changes in this tissue were very noticeable. The alterations in both musculature and elastica are viewed by the author as a degenerative process dependent in all probability upon the typhoid infection.

Experimental Measles.—HEKTOEN (*Journal of Infectious Diseases*, 1905, vol. ii. pp. 238–255) reviews the various attempts that have been made to produce measles by inoculation, and concludes that there is no absolute proof that any of these attempts have been successful. His own work upon the subject consists in two apparently successful attempts to reproduce the disease by human inoculation. In 2 cases typical measles occurred thirteen days after injection into young adults of blood withdrawn from patients suffering from measles. The blood was taken during the first thirty hours of the eruption and kept for twenty-four hours in ascites broth at 37° C. before inoculation. All precautions against contamination of the blood were carefully observed, and the patients in whom the inoculations were made were free from exposure

to measles, both being convalescent from scarlet fever in hospitals that contained no cases of measles. The author considers that his experiments show that the virus of measles may be obtained unmixed with other organisms and in such a form that it may be studied by various methods.

Immunization against Tuberculosis.—BAUMGARTEN (*Verhand. der deut. Path. Gesellschaft*, 8th Tagung., 1904, Heft 2, p. 133), stimulated by v. Behrring's observations upon the apparent immunization obtained in cattle against the bacillus of Perlsucht by previous inoculations of slightly virulent human tubercle bacilli, repeated the experiments in the hope of discovering a means of protecting man. He found that cattle treated with human tubercle bacilli acquired an immunity against doses of the bacillus of Perlsucht which were fatal to control animals. This immunity may last for two and one-half years. Even a single subcutaneous injection of human tubercle bacilli will protect against Perlsucht infection, though the animal remains immune not longer than a year. The injection of bacilli from human sources, highly virulent for guinea-pigs, does not produce in the cattle a typical local tuberculosis; the tubercle bacilli are rapidly destroyed and at the points of inoculation only inflammatory changes are found. These experiments, according to Baumgarten, do not oppose but rather substantiate Koch's doctrine of the duality of human and bovine tuberculosis; the blood serum of these immunized animals has not, up to the present time, possessed either protective or curative properties for rabbits and guinea-pigs. In case the future is unable to offer a curative serum for tuberculosis in man, the possibility of a protective immunization similar to that obtained in cattle may be considered.

In this connection, the experiments of Pearson and Gilliland (*University of Pennsylvania Medical Bulletin*, 1905, vol. xviii. p. 30) are of great importance. In testing a herd of shorthorn cattle, which were known to be tuberculous with tuberculin, twelve calves from six to eight months old, were found which responded to the test. These animals were divided into two lots of six each, as nearly equal as possible in respect to age, size, weight, and conditions. One of these lots was then subdivided into two groups of three each. The three calves of one of the subgroups were given seven intravenous injections of a standard suspension in water of tubercle bacilli of human type, the inoculations covering about three months. The three calves of the other subgroup were given after each injection of human tubercle bacilli from three to five injections of tuberculin (until the hypersensitiveness to the tuberculin was lost). The second group of six calves were kept as controls and were given no treatment whatsoever. All the animals were kept together under exactly the same conditions.

Two controls died before the experiment was completed, one control and one treated calf were killed about one year after treatment, and the remainder were killed at the end of eighteen months. All the six control or untreated animals showed more or less widespread and active tuberculosis; in one animal the disease was in the most advanced form and of widest distribution. In all of the six treated animals the lesions were small, quiescent, and encapsulated, though they contained living tubercle bacilli. The observations thus showed a decided difference between this lot of young cattle that were treated and the six that were not treated. Since the two lots of animals were in all respects as nearly

equal as possible at the beginning of the experiment, and since they were cared for all together in precisely the same way, excepting in respect to specific treatment, the authors think it but fair to conclude that the six treated animals were favorably influenced by the treatment. The authors hold that under such treatment as was given, tuberculous lesions do not extend, on the contrary, that they recede and that new implantations do not occur, even upon prolonged contact with tuberculous herd mates.

The Biology of the Micro-organism of Actinomycosis.—J. H. WRIGHT (*Journal of Medical Research*, 1905, vol. xiii. p. 349) presents in an essay, to which was awarded the Samuel D. Gross Prize of the Philadelphia Academy of Surgery, an extensive study on the biology of the branching filamentous micro-organisms isolated in pure culture from 13 cases in man and 2 in cattle of actinomycosis. He considers the organisms to be all of one species, essentially an anaerobe, growing well only in agar and bouillon and in the incubator. He found that the filaments took on the "club" appearance only when grown in immediate contact with animal fluids or within the animal body. Experimental inoculation caused the formation of tumor masses of small sizes, which showed but little progressive tendency. The organism could be regained, but there was little evidence that multiplication had taken place within the animal.

The author considers actinomyces bovis to be the proper name for the organism. He does not accept the prevalent belief, based on the work of Bostroem, Gasperini, and others, that the specific infectious agent of actinomycosis is to be found among certain branching micro-organisms widely disseminated in the outer world, which differ profoundly from actinomyces bovis in having spore-like reproductive elements. On account of the fact that the organism he described does not grow on all culture media and practically not at all at room temperature, the author does not believe that it has its usual habitat outside of the body. He advances the hypothesis that the organism is a normal inhabitant of the secretions of the buccal cavity and of the gastrointestinal tract. The part played by foreign bodies he believes is in the formation of a nidus where the actinomyces develop to form actinomycosis, and he does not think that straw and like foreign bodies so frequently found in actinomycotic lesions are the carriers of the micro-organism.

The "club" formation about the filaments is considered as a protective function. The bacteria so frequently found accompanying the disease are, the author believes, important in the spread and continuance of the infection, forming new sinuses to be reinfected with actinomyces bovis from the buccal cavity and the gastrointestinal tract.

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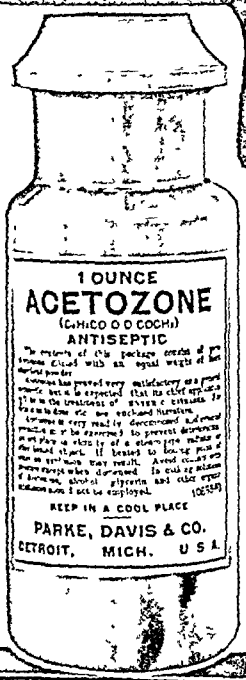
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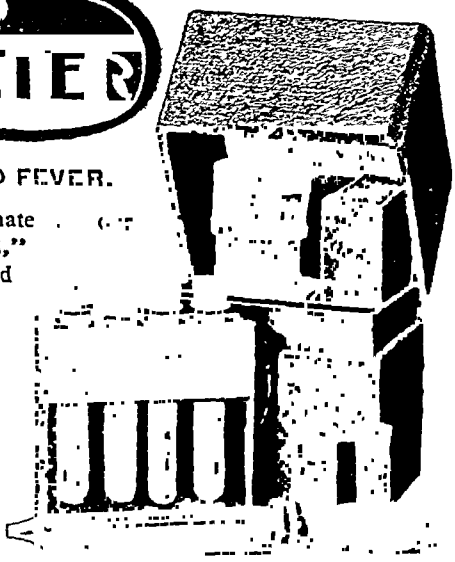
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AUGUST, 1905.

NOTES ON OTITIC EPILEPSY.[1]

WITH REPORT OF A CASE RELIEVED BY MASTOID EXENTERATION.

By B. Alex. Randall, A.M., M.D.,
of Philadelphia.

The broadening of the field of aural surgery and the increasing frequency of intervention for intracranial conditions has greatly enhanced the importance to the otologist of the neurological side of this subject, and has made all convulsive and paretic phenomena matters of greater importance. Pathological research is only slowly aiding clinical study in distinguishing between the various affections in which aural and intracranial symptoms are simultaneously present. The epileptiform seizures ascribed to ear disease, especially to plugs of cerumen or other foreign bodies in the external auditory canal, are mentioned by all of the older writers, as Troeltsch, Toynbee, and Wilde; but have been less noted in the newer books. Many of those reported seem hysterical rather than epileptic; and in others, such as several of my own cases, the ear disease in an epileptic has not necessarily had any relation of cause or effect. One case I reported to this society in 1891, and another with rare attacks in which operation has as yet shown no unmistakable influence need not be here discussed; still less those conditions which have been purely incidental, as in the case which I reported,[2] where bilateral hæmatoma of the lobule was caused in an epileptic by the peculiar reviving manœuvre of her father, who lifted her by the ears. One recent case has seemed, however, very clearly otitic in origin and maintenance, and while insufficient time has yet elapsed to permit the operative interference to be properly accredited with a full *cure*, there is at least a promising outlook in that regard which seems to justify reporting it at this time.

[1] Read before the American Otological Society, Boston, May 10, 1905.

[2] Archives of Otology, vol. xxiii., March, 1894.

CASE.—A boy, aged eight years, of American parentage, and apparently in good health, received a drenching by a hose with a penetration of water into his left ear, in the early part of August, 1904. Acute suppuration followed, and, as it slackened, epileptiform attacks, sometimes four or five in the day, began about September 1st. He had suffered "gatherings in the head" as a baby, but had been free from recognizable trouble ever since. He was brought by Dr. Dwight, of Smyrna, Delaware, to Dr. James Hendrie Lloyd, in Philadelphia, and by the latter admitted to the Methodist Hospital on September 13th. His pulse, temperature, and respiration were about normal, the urine and bowels in good condition; but the leukocyte count was 18,000 on the 17th, 11,400 on the 19th, and 8000 on the 20th and 21st. Other causation of his attacks not being evident, his ear condition, with moderate discharge of yellow, odorless pus, led Dr. Lloyd to call me into consultation, and I examined him on the 21st, after he had been waiting about a week for my return to the city. The right ear was found free and fair. On the left the fundus of the canal was filled by a polyp-mass attached up and forward by a base too broad to admit of torsion. This was largely removed, but its attachment was fibrous and tough, and bare bone could be felt beyond it. Exploration, and probably exenteration, was arranged for the next day under ether. The eye-grounds were fair, yet with the nerve margins much veiled by retinal striation; but there was no swelling of the disk or notable tortuosity of the vessels.

Precise notes as to his seizures were not at first secured. He had an attack of rigidity in his admission bath, lasting nearly two minutes; but slept fairly his first night in the hospital, with only some gritting of the teeth. Next morning there had been twitching of the right thigh and leg and of the left hand, but no other attack upon the 14th. On the afternoon of the 15th, he had suddenly become rigid while sitting up in bed, sinking slowly down to a recumbent position by reason of straightening of the back, winking both eyes rapidly, especially the right, and seemed unconscious for about sixty seconds. He cried after it had passed, complained of a slight headache, and seemed to be much disturbed by his mother's emotion. This was followed by a good night, with some leg-twitching and gritting of the teeth. On the 16th there was a slighter attack in the afternoon, and he noticed some pain in the ear that night and the next morning, when he had another well-observed attack, in which the whole body and limbs stiffened suddenly, the left elbow flexing strongly, with the third and fourth fingers bent toward the palm, while the others remained nearly straight. Winking was marked as before, but the right eyelid moving two or three times as rapidly as the left. There was no pallor of the face, but he seemed unconscious during the sixty seconds of the attack; yet he went on playing undisturbed as soon as it was over. He told of a later attack this day not observed by the attendants. On the 18th there were

two attacks in the afternoon, in which he bent slowly backward, with both arms extended straight in front of him and the right index and middle fingers tightly flexed. The attack terminated with the usual relaxation and a sinking down in bed; so he was kept in bed for safety's sake, and laughed and talked unmoved as soon as consciousness returned. There was a spell in the forenoon of the 19th, and two in the afternoon. On the following morning, while kneeling in bed, he had a seizure and fell on the floor without sustaining any injury. He had another attack while eating dinner, and a third a couple of hours later. On the 21st he had a seizure while standing up in bed, but grasped the foot-rail and did not fall. On the 22d he had an attack in the afternoon, and was operated on under ether at 3 o'clock. Caries of the ossicles and of the adjacent wall of the tympanum was found in exploring through the enlarged opening of the Rivinian region; so tympanic exenteration was carried out and the attic and antrum curetted smooth. The ossicles were markedly carious, and granulation tissue occupied the antrum; but, as there had been no evidence of mastoid involvement and the bone felt firm in that direction, the operation was limited to the tympanum. The tegmen seemed intact, without any baring of the overlying dura. Slight shock followed the operation, with a rectal temperature of 97° and pulse 98, followed by a little rebound. The course of his case was uneventful, the temperature generally normal, although once reaching 99.4°; but the sutured posterior wound refused to heal securely by prime intention at its middle portion, and redundant granulations protruded at this point. On October 11th he was transferred to the Home for Crippled Children, with conditions remaining much the same, and suffering one or more epileptiform attacks nearly every day. The dermatization of the open tympanic cavities proceeded fairly, but the mastoid wound remained open for a week more. He was seen in consultation by Dr. W. W. Keen, who concurred in regarding the aural condition as the cause of his seizures, and advised expectancy until the completion of the healing after the exenteration. The protruding granulations were shaved away from the mastoid wound, and firm healing then ensued, and he was sent home the last of October with his ear almost dry and dermatization apparently completed. The physician at home, Dr. Dwight, reported continued discharge, with granulations on the back wall of the canal. The patient reported at my office on November 23d, showing exuberant granulations on the back wall of the canal, which were curetted away and seemed to be controlled; but when seen again on the 25th the suspected sinus was demonstrable at this point, and at a second consultation with Drs. Keen and Lloyd further operation was decided upon, with possible trephining in the motor region of the left side if insufficient finding in the mastoid region left the matter of cure in doubt. Complete exenteration of the mastoi was done December 1st, at the University Hospital, finding unh-

bone and granulations in the mastoid, but its inner depth apparently everywhere sound. The lateral sinus was laid bare without finding granulations or pus in the sulcus, and the tegmen removed from the attic and antrum with like negative finding.

During the interval his attacks had been even more numerous than before, the minor tonic convulsions being reported as sometimes twenty in a night, as well as repeated and severe in the day. At the University Hospital my Resident, Dr. Gray, kindly spent an entire night noting the frequency and symptoms of his seizures. These attacks usually took the early form of rigidity and curving of the back, with the arms stiffly outstretched in front, but later one or both arms were raised at times vertically above the head. The wound healed kindly, and he was transferred to the care of Dr. Dwight, in Smyrna, on December 16th, with little cavity yet to fill up with granulations; and in another two weeks the healing was complete and sound, with dry ear, hearing not greatly reduced, and his mastoid region free from tenderness. When last seen on February 23d, all seemed in good condition, and his attacks had markedly decreased in frequency and severity. After that he had but two attacks at a fortnight interval, and had been a month without any occurrence whatever. The latest report from Dr. Dwight (April 14th) is to the effect that "the boy seems entirely cured."

Schwartze's case of epilepsy relieved by mastoid trephining is almost the only instance closely paralleling the case here reported, and the completeness of the result must in his record be inferred rather than distinctly found. Most of the other cases bearing upon the subject have been much more fleeting in the onset and more easy of relief, through the cure of pus-retention, Eustachian stenosis, or the irritative presence of cerumen or foreign body in the canal. Some of these cases seem really hysterical; in others the ear condition has been but an incident in an epileptic patient, and relief has been experienced as so often follows every intervention of whatever sort in such patients. Where this has been the result of a single inflation, the removal of a wax plug, or the incision or perforation of the drumhead with evacuation of pent-up secretion, it would seem a magnifying of the conditions to call the case one of otitic epilepsy, or to claim a cure from what has been followed by relief. It is different, however, where the presence of a polyp or of an irritative foreign body has been accompanied by epileptic convulsions for months or years, and relief has followed the abatement of the ear condition.

Two such cases from polyp as well as pus are reported by Dalby; of others, Mendoza's case is more frequently quoted. A verbal report from Christian R. Holmes notes apparent cure by relief of Eustachian stenosis in two well-established cases: one in a man of thirty [illegible] as others have reported the occurrence of epileptic seizures

at the time of penetrating a stricture of the tube. Urbantschitsch reports cases of what he terms cortical epilepsy, but relief by single inflation marks the case as having been rather trivial, and Gradenigo well sums up in Schwartze's *Hand-book*, when he says: "The complication of ear disease in epilepsy must yet be often looked upon as accidental; the treatment of the one modifying in no way the disease process of the other."

REPORT OF TWO FATAL CASES OF BRAIN ABSCESS.[1]

By Edward Bradford Dench, M.D.,
of New York.

The following cases of brain abscess, although ending fatally, seem to me to be of sufficient interest to be reported in full:

I. G., aged fifty-two years, was admitted to the New York Eye and Ear Infirmary on February 6, 1905, with the following history: The patient had always been in good health; no specific history; used alcohol in moderation. A year ago he suffered from pain in the right ear, which disappeared in a few days. One month previous to the patient's admission to the hospital, he was taken with a severe earache. On the following day he consulted a physician, and was under treatment for two weeks, with some relief; there was a little discharge from the ear at this time. At the end of two weeks' time the pain in the ear had ceased, but the patient began to complain of pain in the right frontal region, over the right eye. The patient's family reported that for two weeks before his entering the hospital he had been decidedly worse, that he had alternate attacks of restlessness and drowsiness, but, as a rule, was able to sleep at night. He had only complained of pain in the right frontal region. Some staggering gait had been noticed during this period. The physician who treated him said that, with the inception of the earache, there was considerable swelling over the right mastoid process, and firm pressure in this region elicited pain. This tenderness and swelling had disappeared before the patient came under observation. On his admission to the hospital he seemed to be in much pain, was restless one minute and drowsy the next. The face had a marked septic appearance. The breath was foul and the tongue coated with a brownish coating. When left to himself, the patient seemed to drop off into sleep as though he were under the influence of some hypnotic, although the use of any hypnotic was denied. From this semiconscious condition the patient was easily aroused. On questioning him he said that he felt perfectly well, and he complained of absolutely no pain; there

[1] Read before the American Otological Society, Boston, May 10, 1905.

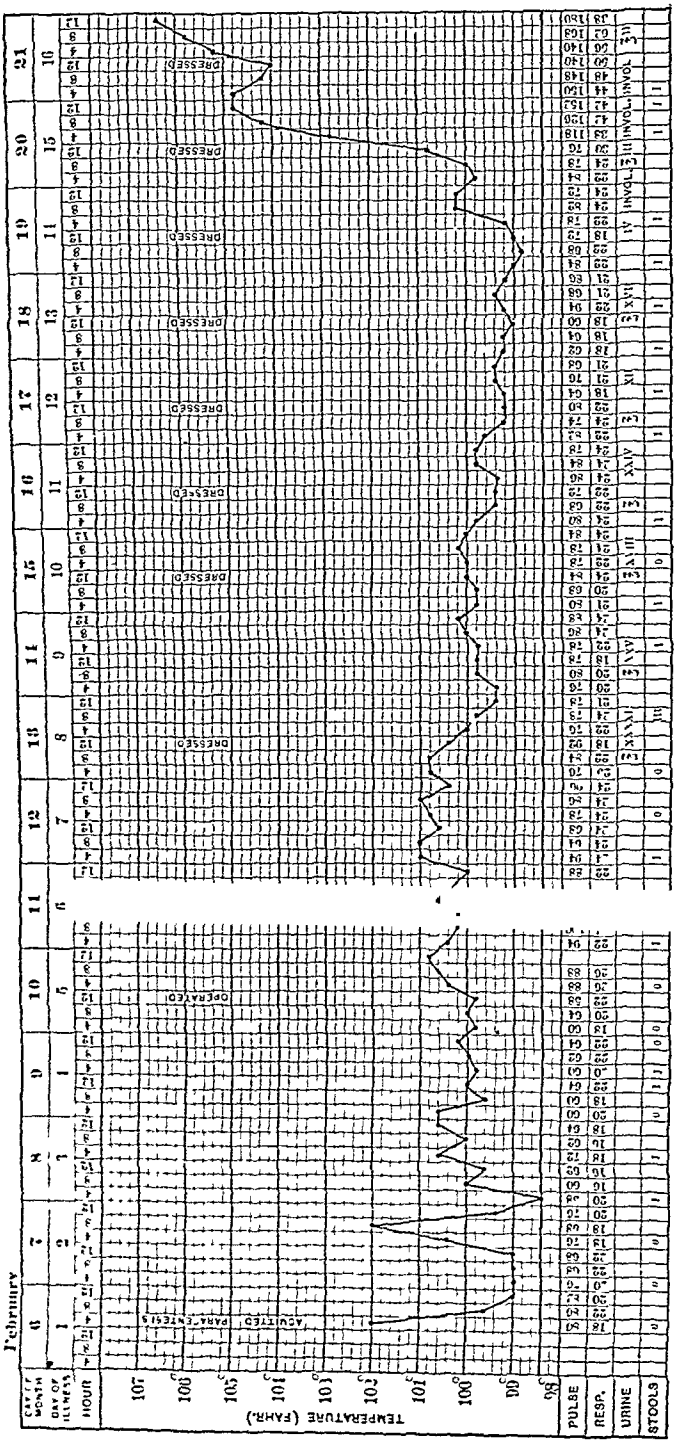

February
TEMPERATURE (FAHR.)
ACUTITED PARATYPHOID
OPERATED
DRESSED
PULSE
RESP.
URINE
STOOLS

CHART I.

was no mastoid tenderness at this time. The right external auditory canal was considerably narrowed throughout its entire extent. There was no marked bulging of the drum membrane, although the upper and posterior portion of the membrane could not be clearly seen on account of the narrowing of the canal. This narrowing was uniform, and was not confined to the fundus.

A free myringotomy, with the extension of the incision on the upper and posterior wall, was performed immediately after admission. A bacteriological examination of the discharge from the ear showed an unidentified bacillus in considerable numbers. General examination showed the reflexes to be about normal; power and sensation, on both sides, normal and equal; patellar reflex slightly exaggerated upon the right side. An examination of the eyes showed the fields of vision and optic disks to be normal. Physical examination of the thoracic and abdominal viscera revealed no departure from the normal standard.

There was practically no discharge from the ear following the incision of the drum membrane. The patient's condition remained about the same for three days. On the fourth day after admission there seemed to be some slight mastoid tenderness, and, owing to the irregular temperature and a leukocytosis, shown on the day of admission to be 21,400, as well as the history of the case, I decided to perform an exploratory mastoid operation. Rather extensive destruction was found in the mastoid, some free pus being present, and the cells being filled with granulation tissue. The lateral sinus was exposed, but was found to be healthy. An examination of the pus from the mastoid wound showed a mixed infection of large and small staphylococci, occurring in pairs, and, in short, irregular chains. Previous to the mastoid operation the patient had been examined by Dr. M. Allen Starr, who found no evidences of intracranial involvement. The day after the operation the patient was more stupid, and there was some paralysis of the left upper extremity. The right arm was moved constantly, and the left arm was not moved at all.

Owing to the fact that the patient was becoming steadily worse, I decided to do an exploratory craniotomy. The cranial cavity was opened an inch and a quarter above and an inch and a quarter behind the centre of the external auditory meatus, and an incision forward, downward, and inward, extending to a depth of about an inch and a half into the brain substance, evacuated about an ounce of pus. The abscess cavity was explored by means of the finger, and was then examined by means of the encephaloscope. The cavity was thoroughly dried through the encephaloscope by means of the cotton-tipped probes, and the entire abscess cavity packed with sterile gauze.

On the day after the operation the patient was able to move his left arm, and seemed brighter that afternoon than he had been at

any time since his admission to the hospital. This improvement lasted for four days, when the patient became dull, and was aroused with difficulty. The abscess was dressed daily, the packing being renewed after the entire cavity had been cleansed thoroughly through the encephaloscope. From the fourth day until the ninth day after the operation the patient steadily became worse; he became more and more drowsy, the temperature gradually rose, and he died, on the tenth day after the operation, with symptoms of meningitis.

The second case was that of a young man aged twenty-one years, who presented himself at the New York Eye and Ear Infirmary on December 5, 1904, with a history of having had a constant purulent discharge from the left ear during the past two years.

On examination, a slight purulent discharge was found in the canal; the membrana tympani was very extensively destroyed; no granulation tissue was present. The radical operation was performed in the usual way. The antrum was very deeply located, and was also situated at a higher level than normal. Considerable softened bone was found in the epitympanic space, and owing to the amount of destruction and to the rather free hemorrhage during the operation, primary grafting was not deemed advisable. After the ordinary conchal flap was cut, the posterior wound was packed with iodoform gauze and allowed to remain open. A secondary skin grafting was done two weeks later, the granulations which had sprung up in the bony cavity being removed by means of the curette at the time of the grafting. Two large Thiersch grafts were applied to line the cavity, and the posterior wound was completely closed by sutures. The temperature began to rise very soon after the grafting, and three days after the secondary operation all pledgets were removed and the grafts taken away. In spite of this fact, however, the temperature four days after the secondary operation had reached 105.6°. At the dressing on the previous day the wound was found to be infected. All stitches were, therefore, removed, and the wound irrigated freely with a peroxide of hydrogen solution. The temperature then fell gradually for five days, until it reached 99.2°, and the patient seemed to be doing very well. At the time of the high temperature the blood count showed 25,000 white cells, 6,000,000 red cells, 81.5 per cent. polynuclear cells. As the temperature fell, the leukocytosis promptly disappeared, so that on December 27th, when the temperature had reached 99.2°, the leukocytosis had fallen to 13,000. The patient continued to improve, and three days later the leukocytosis had fallen to 11,000. The patient did well for about two weeks, when he gradually became dull, and the temperature began to rise slowly. He complained of considerable pain in the head and some pain in the back of the neck. An examination of the eye-grounds, on previous occasions, had been entirely negative. On January 11th, however, there was decided choking of the left optic disk and the patient

CHART II.

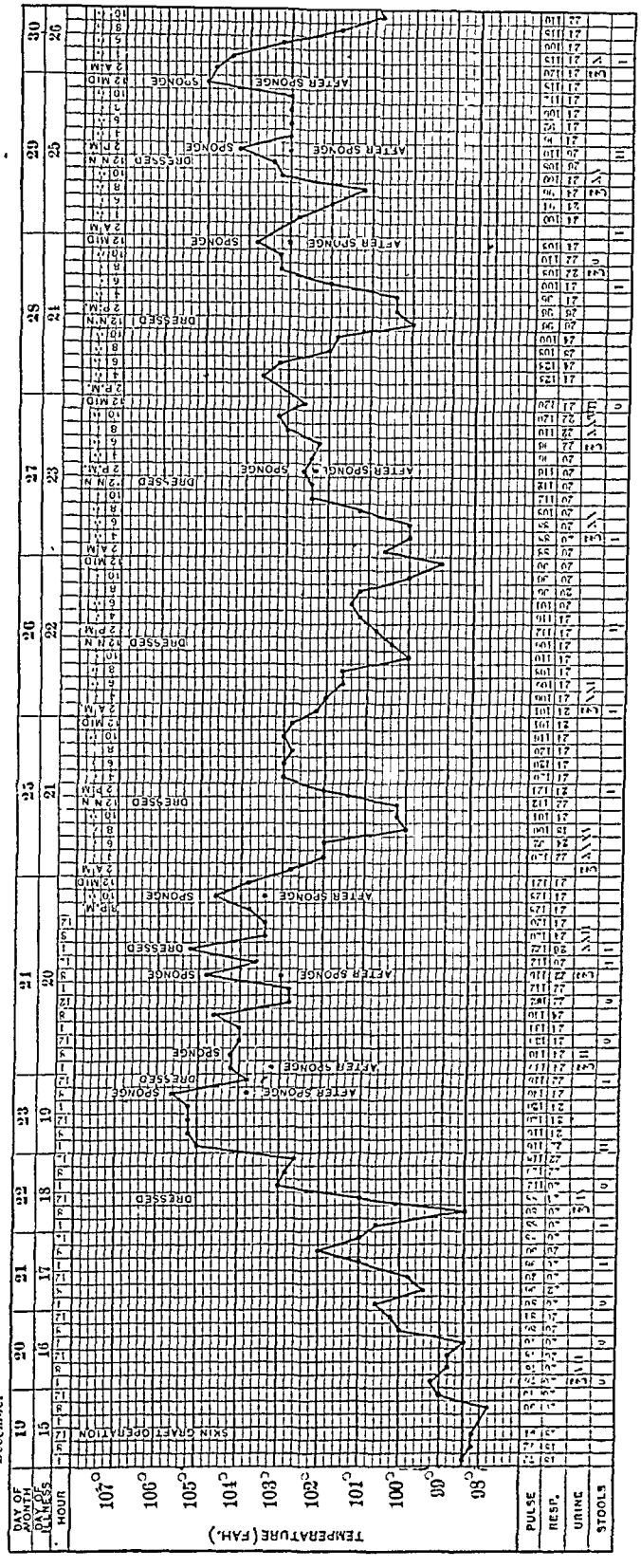

December
TEMPERATURE (FAH.)
SKIN GRAFT OPERATION
DRESSED
SPONGE
AFTER SPONGE
PULSE
RESP.
URINE
STOOLS

CHART II. (Continued.)

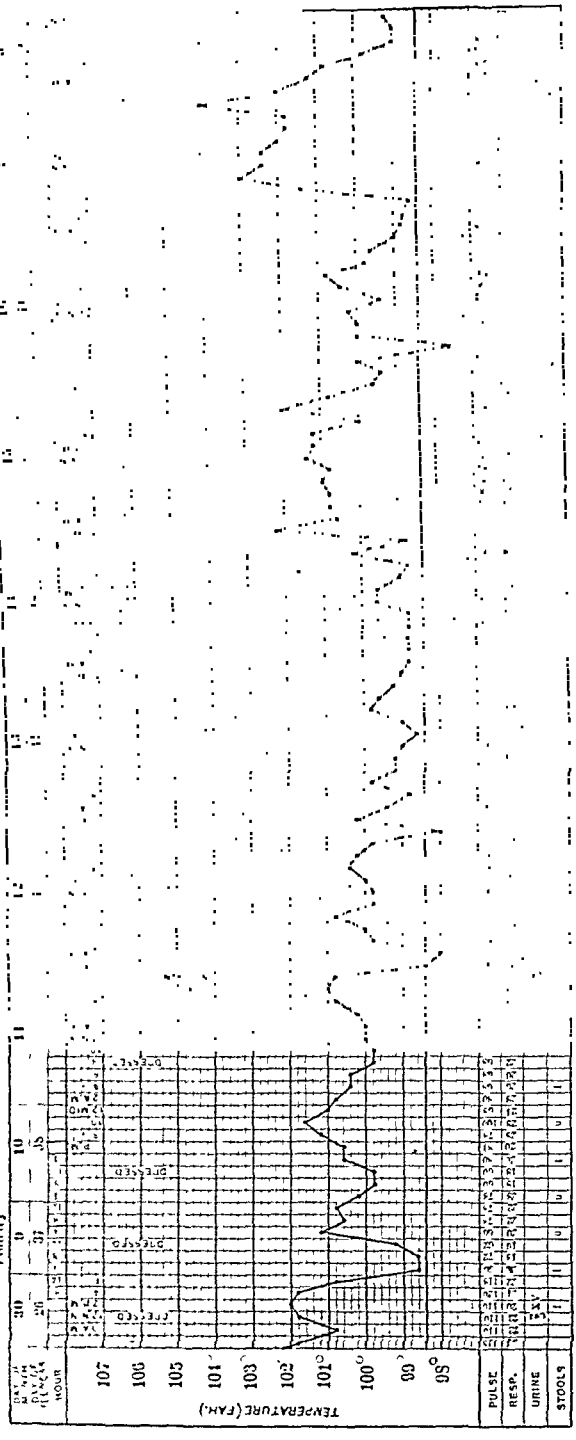
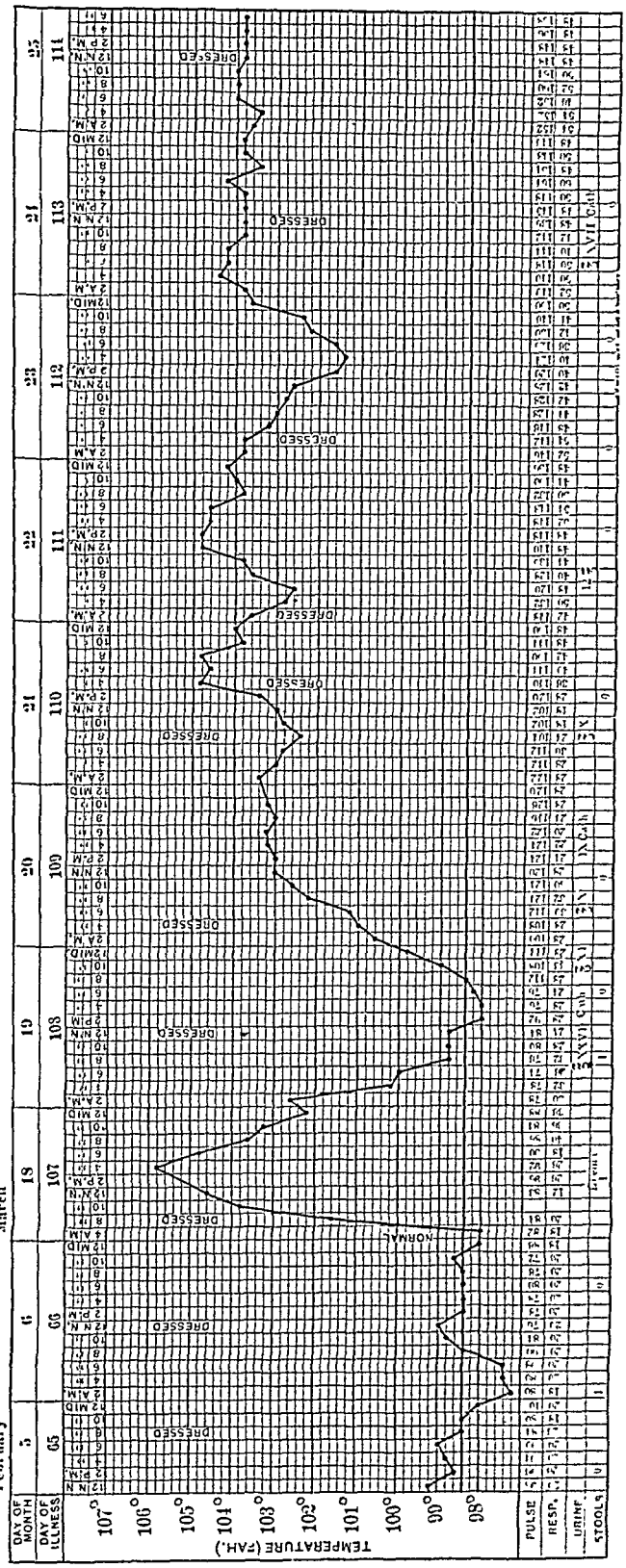


CHART II. (Continued.)



was somewhat aphasic, that is, he could not name, properly, articles which were exhibited to him. For this reason it was deemed wise to explore the temporosphenoidal lobe. A blood count at this time showed 16,500 white cells, 5,400,000 red cells; a differential count was not made at this time. An examination of the field of vision, at this time seemed to show that there was homonymous hemianopsia, involving the temporal half of the right eye and the nasal half of the left. An exploratory craniotomy was therefore performed, and a large portion of the squamous bone removed. A dural flap was then reflected upward and several punctures were made in the brain substance, with negative results. The dural flap was accordingly partially sutured over the exposed cerebral substance and the angles of the incisions in the soft parts were united by means of sutures; the incised brain tissue was dressed with iodoform gauze and separated from the radical operation cavity by iodoform gauze packing. The bandage was then applied and the patient returned to bed.

On the third day after the operation the wound was dressed and the sutures removed. About five days after the operation a small abscess formed under the anterior angle in the soft parts. This was evacuated, and the temperature, which had risen slightly, again fell to near the normal standard. About ten days after the exploratory operation, on examining the hernia cerebri, which was quite prominent, considerable pus was evacuated from the brain substance. There seemed to be two pockets in the brain substance—one extending downward, forward, and inward, and the other extending inward and slightly backward. These pockets were packed with sterile gauze, and the patient continued to do very well. He gained strength, the temperature became normal, his aphasia became less marked, and he was able to be up and about the ward. The hernia cerebri never completely disappeared, although, as the result of firm pressure and repeated excisions, it became considerably reduced in size. A small hernia, however, of an area of about the size of a twenty-five-cent piece still remained. The margins of this were excised, and an application of nitrate of silver made to the cut margins. Firm strapping of adhesive plaster was also applied over the hernia, to reduce its size.

From the time of the operation upon the brain, on January 11th, until March 18th, the patient continued to improve. There were some slight temperature remissions, but, with one or two exceptions, occurring within the first week after operation, there was never a rise above 100°, except on February 4th, when, for some unexplained reason, the temperature suddenly rose to 104°. From February 4th the patient continued steadily to improve. He gained strength, the aphasia almost entirely disappeared, and although his vision was not perfect it had improved considerably. He was able to be up and about the ward, and was anxious to leave the

hospital. All of these favorable symptoms continued until March 18th, when he complained of severe pain in the head, had a chill, and became comatose, the temperature rose to 105.8°, the neck became rigid, and he presented all the evidences of meningitis. The brain substance was again opened over the region of the *hernia cerebri*, and a considerable quantity of turbid fluid evacuated. This fluid evidently came from the lateral ventricle. Immediately after the evacuation of the fluid the temperature fell to normal, but very soon rose again, and the patient died on the seventh day after the invasion of the ventricle.

These two cases present points of considerable interest.

In the first case we have a history of an acute middle-ear inflammation, with the probable involvement of the mastoid at the time of the acute invasion, and with the apparent subsidence of all mastoid symptoms for a period of two weeks. The patient then comes under observation, with ill-defined cerebral symptoms, but with no evidences either of severe middle-ear inflammation or of mastoid involvement. The operation upon the mastoid was only considered justifiable after the patient had been under observation four days, and was then performed as an exploratory measure, as there seemed to be no other reason for the patient's temperature elevation and leukocytosis. Mastoid tenderness was either absent or only present to a very slight degree. The condition of the mastoid at the time of operation and the increasing mental dulness were the indications upon which the operation was performed on the following day on the brain, and, as already detailed, this operation revealed a large temporosphenoidal abscess. As the disease, in this case, was located upon the right side, all localizing symptoms were wanting, until the paretic symptoms, referred to the opposite side of the body, made their appearance.

Whether an earlier operation would have saved this patient is a question. Considering the fact that he was examined by a prominent neurologist only forty-eight hours before I decided to do an exploratory craniotomy, and that, as the result of this consultation, operation was advised against, it would seem that any earlier operative procedure would hardly have been considered justifiable.

In the second case the high temperature immediately after the skin grafting led us to suspect at first a central pneumonia as the result of the ether anæsthesia. Repeated examination of the chest, however, failed to reveal any signs of consolidation, and, as will be seen by referring to the chart, the temperature gradually fell, until it became almost normal. The cerebral symptoms did not make their appearance until about three weeks after the secondary grafting of the middle-ear cavity. In this case the cerebral symptoms were rather complex, and seemed to indicate a somewhat extensive involvement of the brain substance; that is,

the patient was aphasic, or suffering from "word blindness," which would naturally lead one to locate the lesion in the posterior portion of the temporosphenoidal lobe. In addition to this symptom, however, the appearance of homonymous hemianopsia seemed to show either involvement of the occipital lobe or of some of the commissural fibres. Owing to the wide separation of the two cortical areas involved, and presuming that the primary seat of the infection was the middle ear, it seemed wise to me to open the skull immediately above the external auditory meatus, rather than farther back over the occipital lobe. Naturally, in a case of this kind there may be multiple purulent foci. Failing to find pus in the temporosphenoidal lobe, I did not feel justified in exposing the occipital lobe at this same operation, and the steady improvement of the patient after the exploratory craniotomy seemed to rather argue against any further exploratory operation.

I feel that if in this case the opening into the cranial cavity had been made farther back, at the posterior extremity of the horizontal limb of the fissure of Sylvius, the operation might have been more successful. From the fact that pus was obtained from the brain substance a number of days after the operation, although none was obtained at the time of the operative procedure, it seems quite possible that the pus slowly worked its way along the lines of least resistance, and was evacuated spontaneously through the *hernia cerebri*. That the drainage was insufficient is clearly shown by the subsequent fatal outcome of the case.

If there is one thing which the otologist should remember more than another, in dealing with these cases, it is, I think, to attempt in every instance to open the cranial cavity as nearly over the site of the collection of pus as possible so that the abscess may drain through a minimum amount of healthy brain tissue. I think that too frequently we undertake an exploratory craniotomy without having clearly in our minds the exact locality which is involved in the purulent focus. In most of the operations I have performed upon the brain, as well as in those which I have seen performed by other otologists, the general rule of opening the cranial cavity just above the external auditory meatus has been followed, irrespective of the symptoms complained of by the patient. A closer study of the symptoms presented by each individual case, thus enabling us to locate the situation of the abscess in the brain substance, and a more exact knowledge of craniocerebral topography, so that we shall make our opening through the skull as nearly over the affected brain area as possible, will, I think, materially improve our statistics in operations for brain abscess.

For the mapping out of the various fissures and convolutions of the brain, perhaps the lines suggested by Watson Chiene are the best. These lines are as follows: A line is drawn from the glabella over the top of the head to the external occipital protuberance,

and three points are taken on this line: the midpoint, the three-quarter point, and the seventh-eighths point. A second line is drawn from the external angular process of the frontal bone to the preauricular point, that is, the root of the zygoma. From the external angular process of the frontal bone a line is drawn to the three-quarter point of the original line and from the root of the zygoma a line is drawn to the seven-eighths point of the original line. The midpoint of the line between the external angular process of the frontal bone and the preauricular point is then taken, and also the midpoint of the line from the preauricular process to the seven-eighths point, on the first line drawn over the top of the head. The three midpoints are then joined, that is, the midpoint of the line running from the root of the nose to the occipital protuberance and the two midpoints just mentioned. The point where the anterior line joining the midpoints cuts the line drawn from the external angular process of the frontal bone to the three-quarter point represents the anterior extremity of the fissure of Sylvius, and just above this point lies Broca's convolution, lesion of which gives rise to motor aphasia. Where the posterior line joining the midpoints crosses this same line, that is, the line from the external process of the frontal bone to the three-quarter point, is the area of "word seeing." The area of "word hearing" lies in the superior frontal convolution, just midway between the anterior extremity of the Sylvian fissure and area of "word seeing," and just below the level of the Sylvian fissure. The anterior line joining the midpoints corresponds in its upper portion to the superior precentral fissure. A line drawn from the point already mentioned as indicating "word hearing," and parallel to the precentral fissure, marks very closely the postcentral fissure. The fissure of Rolando runs between the precentral and postcentral fissure, midway between these two. The visual area may be found by drawing a line from the three-quarter point downward and forward, following the line of the parieto-occipital fissure.

Perhaps this method of localization may possess some advantages over the older method described of making measurements from Reed's base line. It is unnecessary to remember any exact figures in drawing these lines, and as the localization is accomplished simply by taking proportionate distances along these various lines joining bony landmarks, the system is equally applicable to adults and to children.

The method of craniocerebral topography devised by Chipault is, perhaps, quite as useful as that of Watson Chiene, and, if the metric system of measurements is used, no more difficult. According to this method a line is drawn from the nasofrontal suture to the occipital protuberance. The points taken on this line are 0.45, which is the pre-Rolandic point, 0.55, 0.70, 0.80, and 0.95. The retro-orbital tubercle is then found, and three radiating lines are

drawn from this—one to the 0.70 point, one to the 0.80 point, and one to the 0.95 point, on the original line. The upper of these radiating lines corresponds to the fissure of Sylvius, while the lower, in its posterior portion, corresponds to the lateral sinus. These radiating lines are then divided into tenths. A point 0.2 back on the anterior extremity of the Sylvian line corresponds to the Sylvian point, that is, to the anterior extremity of the horizontal limb of the fissure of Sylvius. The pre-Rolandic line is drawn from the 0.2 point on the Sylvian line to the 0.45 point on the line drawn from the naso-frontal suture to the occipital protuberance. The lower part of the precentral line corresponds to the vertical limb of the fissure of Sylvius. If a point 0.3 backward from the retro-orbital tubercle, measured along the Sylvian line, be joined to the 0.55 point of the line running from the nasofrontal suture to the occipital protuberance, this line will correspond to the fissure of Rolando.

The various frontal convolutions can be mapped out by dividing the pre-Rolandic line into tenths. The inferior frontal convolution extends upward on the pre-Rolandic line for about 0.3 of its length. This corresponds, of course, to the convolution of Broca. The posterior extremity of the Sylvian fissure lies at a point about one-half the distance backward, measured along the Sylvian line, and corresponds closely to the region of "word seeing."

It will be seen, therefore, that by this method the various fissures and convolutions of the brain may be thoroughly mapped out, so that it is possible for the surgeon to know exactly what portion of the brain he is exploring after the opening has been made. These lines should, I think, be laid down on the skull with extreme care prior to an exploratory craniotomy, in order to avoid unnecessary exploration of the brain substance in searching for the abscess. Of course, where localizing symptoms are absolutely wanting, the surgeon may follow the old rule—of opening the cavity just above the external auditory meatus.

A word may not be out of place in regard to the character of the incision in the soft parts in these cases. While, in writing upon this subject previously, I have ordinarily advocated an extension of the mastoid incision over the ear, well forward toward the external angular process of the frontal bone, with forcible retraction of the edges, so as to expose the bone beneath, I am convinced that even when the abscess is found this method of linear incision is far inferior to turning a tongue-shaped flap upward so as to expose the surface of the cranium over a comparatively large area. The turning-up of such a tongue-shaped flap can be very quickly done, and it shortens the operation and facilitates greatly the subsequent dressings in the case. Where the ordinary linear incision is made, or even the ordinary crossed incisions, the flaps fall together so easily that subsequent dressings are extremely painful, owing to the forcible separation of the soft parts at each dressing. If a

tongue-shaped flap is elevated and the bone removed beneath, we have a perfect exposure of the cranial contents without very much retraction of the flap, and the subsequent dressing of the abscess causes the patient very little pain. If the exploration is negative, and the surgeon desires to close the exploratory wound immediately, the flap is very easily sutured in position.

In exploring these abscesses and introducing drainage I have devised a pair of very light retractors for separating the line of incision in the brain substance, thus giving a view of the deeper portion of the abscess cavity. After the abscess has been opened by means of the director or the knife, as the case may be, the director is allowed to remain in the cavity and one of these retractors is passed along the director until it enters the abscess cavity. The director is then withdrawn and the other blade of the retractor is passed along the first blade as a guide. The separation of these retractors will then show the interior of the cavity. If desired, the encephaloscope may be introduced between the retractors and the retractors removed. The disadvantage of introducing the encephaloscope at first is that it must be introduced somewhat blindly, and may do unnecessary damage to the brain substance. The thin blade of the retractor, however, can be slipped along the director and will not make a false passage. After the first dressing, I think that the encephaloscope is a very valuable instrument to be used in these cases, but I think that the narrow retractors are more serviceable at the time of operation than the encephaloscope.

ORBITAL AND MENINGEAL INFECTION FROM THE ETHMOID CELLS; DEATH; REPORT OF A CASE.[1]

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Nasal sinusitis, on account of the widespread interest which has been manifested for several years by the medical profession in the endeavor to arrive at a clearer understanding regarding the pathology and best methods of treatment, make the subject pregnant with interest to us as rhinologists. In the case which I report for your consideration, aside from the desirability of reporting all cases of intracranial involvement consequent upon accessory sinus

[1] Read at the Eleventh Annual Meeting of the American Laryngological, Rhinological, and Otological Society, Boston, Mass., June 5, 6, 7, 1905.

disease that something may be added to our knowledge of this important class of cases, there are several points of interest.

K. C., male, aged forty years, was seen in consultation on October 13, 1904, and the following history obtained:

Family History. Negative.

Personal History. For about ten years has been troubled with "catarrh," some nasal obstruction, and dropping in the throat. For the past two or three years he had noticed a yellow discharge from each nostril, with increasing nasal obstruction. Contracted colds easily; followed invariably by severe neuralgia and a dull heaviness at the root of the nose. These symptoms would usually be relieved by an increased flow of yellow, thick discharge. For several months the patient had complained of aprosexia, a disinclination to apply himself to his business, depression, and apprehension concerning his sanity. Of this he had several times spoken to his family. His present illness began two weeks prior to my seeing him, with what was supposed to be one of his usual severe head colds. This was followed by quite severe pain at the root of the nose, finally spreading over the entire left side of the face. Two days later there appeared some swelling and œdema at the upper and inner part of the left orbit, which quickly spread, involving both lids, conjunctiva, cheek, and left side of nose. At this time the infiltration and swelling of the lids and orbital tissues were so great that it was impossible to obtain a satisfactory view of the globe. For the next week his general condition grew gradually worse, temperature running from 100° to 103°; pulse from 90 to 115, mental hebetude gradually drifting into stupor and finally mild delirium developed. Proptosis increased, and three days before I saw him there appeared a small swelling over the left temporal region. This slowly increased in size, and at the same time the proptosis, infiltration, œdema, and swelling of the left side of the face and orbital contents gradually diminished. It was at this stage I saw the case and upon examination found the following; patient fairly well nourished, temperature 101°, pulse 100, face flushed, drowsy and cerebration slow and hesitating, responding to commands with a syllabic drawl. Motility of eyes was unimpaired; left pupil reacted sluggishly to light stimulation. Slight amount of swelling of the upper lid and chemosis of the conjunctiva of left eye. On a line with and about one inch posterior to the external angular process of the temporal bone was a smooth, rounded tumor about one and a half inches in diameter. This was not especially tender to pressure, and fluctuation was not detected at this time. On examining the nose I found the following condition: Right nostril—slight turgescence of the inferior turbinate, the middle turbinate pushed well over toward the median by a number of polypi springing from the region of the ethmoid cells, pus appearing in the middle meatus between the polyps. Left nostril—general deviation of the septum

to left, greatest deflection at upper part. Inferior turbinate swollen, middle turbinate pushed well over and crowded against the septum, presenting a deep bluish-red congestion and a swollen, boggy appearance. Springing from the region of the ethmoid and filling the middle meatus were several large polyps bathed in thick pus. With the probe dead bone could not be detected in either nostril. The history and findings in this case left little doubt as to the diagnosis. It seemed clearly evident that he was suffering from an intracranial involvement following an acute exacerbation of a chronic suppurative ethmoiditis. From the fact that this patient was in a small town some distance from the city, and as I had no suitable instruments at hand, I advised his immediate removal to the hospital, where it was my purpose to give drainage to such collections of pus as could be found. He was seen at the hospital next morning, but his condition was so precarious that I did not feel warranted in subjecting him to any prolonged operative proceedure; so satisfied myself with freely incising the swelling over the temporal region, which proved to be a subperiosteal collection of about two ounces of pus. The patient remained in stupor, with muttering delirium, subsultus tendinum, high temperature and pulse, involuntary urination, Cheyne-Stokes respiration, and died twenty-four hours later. After death the wound was examined to determine the path of infection. It was found to have originated in the ethmoid cells, broken through the os planum, stripping the periosteum from the roof of the orbit, extending outward and downward to the external angular process of the temporal bone, and there passed out to form the subperiosteal collection of pus, referred to above. Dead bone could readily be detected in the region of the os planum by passing a probe along this route. My finger in the wound would follow subperiosteally the orbital roof, as far as the sphenoidal fissure. Although no rupture in the periosteum was detected, I think we are justified in assuming that this was the path of infection and that a rupture had taken place at some point with subsequent involvement of the intracranial structures. Our failure to find a rupture does not, in my opinion, alter the value of the evidence that this was the path travelled by the infection. It is, of course, possible that in my examination a break in the continuity of the periosteum may have escaped me, or, again, the area of disintegration may have taken place within the cranial cavity, the infecting material being poured directly into the arachnoid space, with a resulting general septic meningitis.

In studying this case the points of interest are:

1. The fact that this patient died from a meningitis the result of nasal suppuration is of interest as clearly exemplifying the dangers in neglected cases of this character. We need only consult the literature of this subject to be convinced that the reports of fatal cases are rare.

2. The path of infection seems to have been an unusual one, the pus finding its way across the orbital cavity to the external angular process and there leaving it, forming a large subperiosteal collection. Travelling backward, it entered the cranial cavity through the sphenoidal fissue, apparently remaining subperiosteal throughout.

3. The question of diagnosis and prophylaxis is probably of greatest interest, and it is for us as rhinologists to sound the warning note and impress upon our fellow-practitioners the importance of instituting appropriate treatment in all cases of nasal suppuration, before they have reached such extremity. Although fatal cases are rare, it is nevertheless a possibility in every case of suppurative sinusitis.

AN UNUSUAL CASE OF ŒDEMA OF THE GLOTTIS.[1]

By Emil Mayer, M.D.
of New York,
Adjunct Attending Laryngologist to Mount Sinai Hospital, Chief of Clinic Ear and Throat Departments to the Mount Sinai Hospital Dispensary; Former Surgeon to the Throat Department New York Eye and Ear Infirmary; Fellow of the American Laryngological Association, etc.

The patient, a male aged fifty-one years, was admitted to the surgical ward of Mt. Sinai Hospital in the service of Dr. A. G. Gerster, with the diagnosis of dyspnœa.

His history, for which I am indebted to the courtesy of Dr. S. Heiman, House Surgeon, was as follows: Family history negative. Had typhoid at the age of ten. Had a chronic cough with mucopurulent expectoration, never bloody; no night sweats or dyspnœa, no swelling of feet; had never had syphilis or gonorrhœa; is a heavy smoker. Married; the father of two children, both of whom are living. His voice has always been clear; has never had an attack like the present one, but has awakened at night with a sensation of choking, which he attributed to his cough.

His present illness began on April 1st, when he awoke with a pain in the neck on the right side, the attack of dyspnœa coming on suddenly. He rapidly became worse in spite of leeches to his neck and cups to his chest. His voice was that of a whisper, and no relief forthcoming, he was transferred to the hospital April 2d at 4 P.M.

On admission he had severe respiratory dyspnœa and could not lie down; his lips and finger-tips were cyanosed. All accessory muscles of chest are brought into play on inspiration and expira-

[1] Read before the American Laryngological Association at its Twenty-seventh Annual Congress, Atlantic City, June, 1905.

tion, and many moist rales. Respiration 14 per minute. Heart action rapid and forcible. Pulse equal, rapid, and regular; slight tension. Profuse mucus discharge from mouth and throat; breath fetid and the epiglottis could be felt as a thick, hard ridge.

Two hours later, in response to an urgent summons, I saw the patient. He was seated upright in the room adjacent to the operating theatre where he had been conveyed preparatory to a tracheotomy, if such was deemed necessary.

The history given by Dr. A. A. Berg, the adjunct attending surgeon, was that as outlined above. The dyspnœa had been constant for the last thirty-six hours, steadily increasing since his admission.

The expression of the patient was most anxious; he was cyanotic, his breathing very labored with notable inspiratory difficulty. There was a gurgling sound to each inspiratory effort and quantities of mucus were expelled.

The laryngoscopic examination showed a large glistening mass at the base of the tongue, at the right side, filling the entire pyriform sinus on that side and extending over the epiglottis to such an extent as to leave but a narrow opening on his left side for breathing.

The appearance of the mass was that of an œdematous infiltration, immobile on respiratory effort, yielding to the touch of a probe. Except on the left side the epiglottis could not be seen, and an intralaryngeal examination was impossible.

The diagnosis of an acute septic inflammation was made, the parts were freely cocainized, and a deep incision was made with the Hartman laryngeal knife in the presence of Dr. Berg, Dr. Moschowitz, Dr. Libman, and the house staff. A quantity of thick, bloody, and ill-smelling fluid exuded and the patient was returned to the ward.

A spray of adrenalin 1:5000 was frequently applied, subsequently alternating with steam inhalations. His urgent distress was promptly relieved, his breathing became quieter from hour to hour, and all inspiratory difficulty ceased on the second day.

A laryngoscopic examination then showed a large white slough where the œdematous mass had been. A portion of this was removed, and examination showed the presence of staphylococcus albus and streptococcus. His dysphagia also became less and less, the lymph deposit entirely disappeared and in the same place there was now a solid mass of new-growth of firm consistency lying in the pyriform sinus, covering three-fourths of the epiglottis, but not adherent thereto, and which did not interfere with deglutition or respiration. The glands of the neck were not enlarged.

The patient's recovery was rapid, and on April 15th he was discharged from the hospital as cured of his dyspnœa and became an ambulatory patient in my service in the out-patient department of the hospital.

A small piece of the mass was removed with the punch for the purpose of ascertaining the true nature of the growth, and as the report of the pathologist was negative a much larger portion was then removed with the snare, to ensure an examination of the deeper tissues. The report of the pathologist, Dr. F. S. Mandlebaum, was that there was no evidence of anything other than connective tissue in either specimen.

The question of malignancy having been thus excluded, it was evident that this mass was of recent formation and composed of purely inflammatory tissue. Within a short time no further evidence of the growth could be seen and there were no symptoms of any annoyance referable to the throat.

A laryngoscopic examination of the patient made six weeks after the attack showed a most unusual state of affairs as regards the epiglottis. More than half of the epiglottis on the patient's right had sloughed away and a band of adhesion formed running across the lateral pharyngolaryngeal space on that side. The remaining portion of the epiglottis on the patient's left stands erect like an index finger and here two bands of adhesion exist from the epiglottis to the lateral pharyngeal wall. Both of these bands are about one-half inch in length; there is no abnormality within the larynx.

The patient has regained his former health and strength, and bears all the outward appearance of being entirely well.

The point of interest in this case lies in the fact that we have before us a case of œdema of the glottis engrafted upon an hereditary syphilis of the epiglottis, which latter never produced any notable symptoms relative to the throat except perhaps the constant cough of twenty-eight years' duration. The patient recalls that fluids would often come through the nose of late years. The absence of any initial lesion, as also of any evidence of the disease elsewhere, would point most strongly to the hereditary nature of the affection. In fact, the only other condition aside from malignancy that could be present is lupus, and this may be excluded by the absence of tubercle bacilli in any of the specimens examined by the pathologists, and also by the absence of any breaking down of the cicatrized surface, a fact which would occur in all probability immediately after the acute process just ended.

The presence of so large a mass of inflammatory tissue beneath an œdematous process is also remarkable.

This case further illustrates the advantage of prompt, free, and deep incisions into the œdematous tissue, thus avoiding the graver operation of tracheotomy, and emphasizes the value of adrenalin in the subsequent treatment of these conditions, corroborating the statement made in a recent article by Dr. Thomas R. French, of Brooklyn.

SARCOMA OF THE VOMER, WITH EXTENSIVE INVOLVEMENT OF THE ADJACENT STRUCTURES AND METASTASIS IN THE CRANIUM.

By Arthur P. Herring, M.D.,
of Baltimore, Md.

Sarcoma of the nares and nasopharynx is of comparative infrequency. Bosworth in 1899 was the first to publish a series of 41 cases of sarcoma of the nares which he gathered from the literature and from his personal experience. In 1902 J. S. Gibb added 70 cases of sarcoma of the nose and accessory sinuses to those published by Bosworth, making a total of 111 cases reported up to that time. Gibb arranged these histories in tabulated form with reference to special features, and reports 2 cases from his own experience. In 1904 J. A. Watson made "an inductive study based on the records of 150 cases of sarcoma of the nasal passages." Watson's study includes the cases reported by Gibb and a review of the literature up to date. He adds 4 cases from his personal experience.

A careful review of the literature up to April, 1905, shows that there are about 175 cases of sarcoma of the nasal passages reported. That it is a rare affection all text-books agree. In most of the books devoted to the nose and throat but brief mention is made of the subject. For example, Bosworth says: "Sarcoma is by no means frequently met with in the nasal passages, and its literature is somewhat limited."

Kyle states that "sarcoma of the nasopharynx is not of common occurrence." De Schweinitz remarks as follows: "Sarcoma of the nasopharynx occasionally occurs." Looking over the recent annual reports of several eye, ear, and throat hospitals the occurrence of sarcoma of the nose is seldom mentioned. The Presbyterian Eye, Ear, and Throat Hospital of Baltimore, in the report of 1904, gives only 1 case of sarcoma of the nasal septum out of 6125 cases treated for diseases of the nose and throat. In 1903 out of 4083 cases no case of sarcoma and only 1 of fibroma of nasopharynx.

The annual report of the Brooklyn Eye and Ear Hospital for 1904 shows out of 1875 cases, in the nose and throat department, only one of sarcoma of the nose.

The above references serve to illustrate the average frequency of sarcoma of the nares as shown in numerous other reports.

While going over the literature on this subject and reading the discussions of reported cases before medical societies, I was especially impressed with the fact that sarcomatous growths of the nares are of rather frequent occurrence, judging from the remarks made by physicians in discussing the cases reported. I am inclined to believe

that the rarity of sarcomatous growths affecting the nares is only relative and is largely due to the fact that many cases are not reported. The subject is of importance, especially to the general practitioner, from the fact that a neoplasm occurring in the region of the nasopharynx is not always recognized in its incipiency, when it may be removed, thereby offering the patient every chance for complete recovery.

The case reported in this paper is of interest owing to the presence of a metastasis in the cranium, formed early in the development of the tumor, and that the diagnosis of the true condition was obscured for a time, and also the extensive involvement of the surrounding tissue. Concerning the frequency of metastatic deposits Gibb, from his study of 150 cases, remarks that: "Though usually considered one of the most important manifestations of malignancy, the occurrence of metastasis is an extremely rare event. I have found it mentioned but three times. In 2 of these cases the cervical glands were involved (as in the present case); in the other —Warthin's case—there was universal metastasis, including even the formation of a free sarcomatous mass in the right ventricular cavity of the heart." I have been unable to find additional cases from the recent literature. The presence of some intracranial disturbance in this case (either brain abscess or neoplasm) was diagnosed before the growth in the nose was clearly understood; however, after the malignancy of the nasal growth was determined, a metastatic deposit in the cranium was fairly clear. The history of the case and notes from the autopsy record are given in condensed form:

Anamnesis. S. H., male, aged seventeen years, white, farmer.

Family History. Negative.

Personal History. Had measles and varicella during infancy. Has enjoyed good health until the onset of the present trouble. In December, 1903, patient received a severe injury to the right side of the head while chopping wood. He dates all of his present discomfort from this accident. As a result, apparently of the trauma, there developed intense headache, earache, and enlarged cervical glands on the right side. Patient entered the Maryland General Hospital in March, 1904, to be relieved of the above symptoms. The cervical glands were removed, but the pain in the head and ear continued (the glands were said to be tuberculous). After this the patient was treated for pharyngitis and later for a subacute catarrhal otitis media. There was no evidence of a purulent otitis or of a growth in the nasopharynx.

Present Condition. I first saw the patient in June, 1904, when the following notes were taken: Patient anæmic and emaciated; has lost thirty pounds during past three months; facial expression apathetic, cerebration slow; formerly patient's manner was cheerful and active; condition has grown progressively worse. Patient's only

complaint is of headache and earache confined to the right side. Eyes react to light and accommodation; slight ptosis of right upper lid, with possibly a paresis of right facial nerve. Both fundi normal. Hearing normal in left ear. In right ear, bone conduction increased; could hear a watch tick only three inches from ear. Slight congestion and inward retraction of tympanum of right ear.

Speech. There is a peculiar mouthing of words; speaks slowly and indistinctly.

Taste. Normal.

Smell. Anosmia on right side, normal on left.

FIG. 1



A sagittal section of the skull showing the extent of the sarcoma which originated at *X* on the vomer. The shaded area includes the vomer, body of the sphenoid, the basilar portion of the occipital and posterior portion of the cribriform plate of the ethmoid bones. *Y*, within the dotted area, indicates the position of the right lateral mass of the ethmoid, which was also destroyed. *T*, the metastatic growth in the middle fossa, entirely separate and distinct from the tumor proper.

Examination of nose and throat reveals a small vascular tumor attached to the vomer in the right nostril, extending about half-way across posterior nares; no involvement of pharyngeal wall. Growth believed to be a polyp; no microscopic examination made. There is frequent epistaxis from the right nostril, caused by the growth. Heart, lungs, and abdominal viscera normal. Reflexes normal. Sensation for all parts of the body excepting the right side of the face is normal. There could be outlined on the right side of the face an area of anæsthesia corresponding to the distribution of the first and second divisions of the trifacial nerve, extending into the buccal cavity and to the palate and pharynx. The patient has a tendency when walking to fall from side to side. The gait is neither spastic, ataxic, nor cerebellar in character. The temperature ranged

during the past three months from 97° to 98° and pulse from 50 to 90, generally about 60; was full, but not very strong. There has been no elevation of temperature. No chills or vomiting. The urine was normal. The blood examination shows: red corpuscles, 2,500,000; white, 10,000; hæmoglobin, 55. Slight relative leukocytosis, probably the result of repeated hemorrhages. Tongue is coated, white, and furred. The breath is offensive. No tender spots over head or mastoid at any point. No convulsive attacks. The bleeding from the nose is frequent and profuse. There is a constant dripping of mucus and blood.

A tentative diagnosis of either cerebral tumor or abscess affecting the temporal lobe, preferably the latter, was made and the patient prepared for a craniotomy. Under anæsthesia a thorough examination of the nasopharynx was made. The tumor, which a few weeks ago appeared to be a benign, pedunculated growth, had now infiltrated the pharyngeal wall, completely occluding the posterior nares on the right side and extending down into the oropharynx and upward to the base of the skull. Malignant growth with cerebral metastasis was at once suspected and the operation postponed, for the present at least. The patient remained in the hospital only a few weeks longer, and then returned to his home in the country. In September, 1904, the patient returned to the hospital, complaining of pain in the head, difficulty in swallowing and breathing, with frequent attacks of epistaxis. Patient is very much emaciated; there is a pronounced bulging of the right cheek and a slight exophthalmos of right eye. Paralysis of third, fourth, and sixth nerves supplying the right eye. The soft palate is depressed and a large mass can be seen filling the nasopharynx and completely occluding the right nostril.

The tumor has increased in size since June, thus making the patient's condition distressing in the extreme. Only liquid diet can be taken. Owing to the patient's deplorable condition and as a possible means of alleviating his suffering, but with no idea as to the permanent cure of the affection, a resection of the right superior maxillary was performed by Dr. Blake on October 20th. The patient stood the operation very well. A large cauliflower growth was removed from the nasopharynx. The superior maxillary bone was not involved, but the nasal septum and the sphenoid sinuses were destroyed; the tumor mass could be easily scraped away with the finger. The wound was closed, the patient removed to the ward, and lived for about six hours. The autopsy was performed two hours after death. Only the notes referring to the head will be given, as there was nothing of interest in the remainder of the examination.

On removal of the calvarium, which is very thin and translucent over its entire extent, the dura mater being nowhere adherent, the brain is found to be anæmic, the convolutions small and irregular,

especially over the frontal lobes. After removing the brain a smooth, firm, and rounded growth about the size of an English walnut is seen occupying a part of the right middle fossa of the skull, placed close to the apex of the petrous portion of the temporal bone on its anterior surface. This growth is beneath the dura and entirely separated from adjacent structures. Section of the brain presents a normal appearance. In the median line and occupying quite an extensive area

FIG. 2



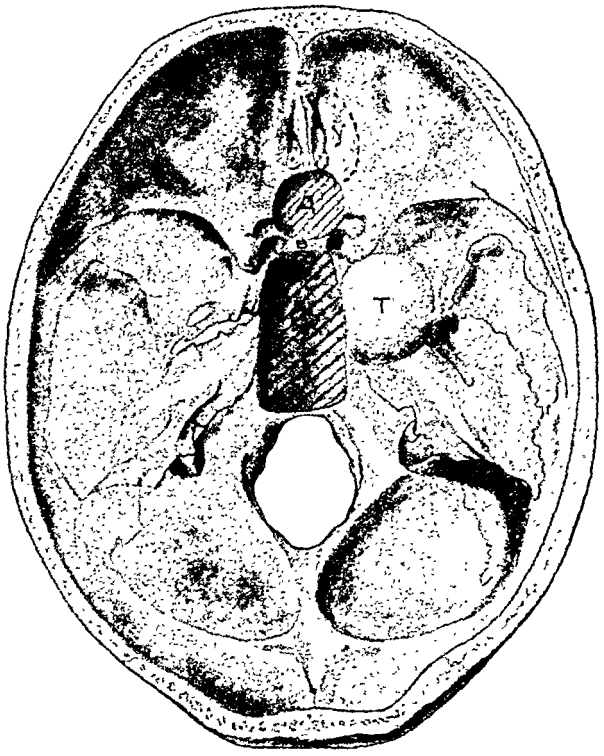
A photograph of the interior of the base of the cranium. The anterior, middle, and posterior fossa are well shown. The dura has not been removed, consequently the entire extent of tumor invasion cannot be seen. The tumor mass in the median line is clearly pictured. The rounded metastatic growth in the middle fossa can also be readily seen. It is firm and nodular in consistency, while the central portion of disintegrated bone is very soft and pulpy. The bony structure has been completely destroyed.

is seen a large, cheesy mass, which will be described in detail. The metastatic growth is separated entirely from the broken-down mass. The anterior surface of the pons is flattened, but not eroded. The incision made during the operation was opened and a thorough investigation of the nasopharynx made, showing an extensive infiltration of pharyngeal wall with complete disintegration of nasal septum and sphenoidal and ethmoidal sinuses. The finger could very easily be pushed from the nose into the cranial cavity through

the disintegrated sphenoid bone. The notes dictated at the autopsy, showing the extent of the neoplasm, are as follows:

The growth originated from the vomer, in the right nasal cavity, and formed a metastasis in the right middle fossa. By direct invasion the tumor destroyed the vomer, passing upward and backward, disintegrating the body of the sphenoid, and filling the sphenoidal sinuses with a creamy matter; then extending posteriorly to the

FIG. 3



A schematic drawing of the base of the skull. *A* represents a circular opening in cribriform plate of the ethmoid bone caused by direct invasion of the neoplasm. *B*, the bridge of bone, optic groove, which was not destroyed. *C*, a large extent of disintegrated bone the result of direct growth of the tumor from the nose. There is a direct communication between the interior of the cranium and the nasal cavity. *T*, the metastatic growth which pressed upon the third, fourth, sixth and the first two branches of the fifth cranial nerves. *Y*, within the dotted area, indicates the inner wall of the orbit which was destroyed causing exophthalmos.

basilar process of the occipital bone, destroying it as far as the margin of the foramen magnum; passing anteriorly, the neoplasm invaded the anterior and superior surfaces of the sphenoid, the os planum, and right lateral mass of the ethmoid, destroying the posterointernal wall of the orbit, thus causing an exophthalmos. A rather remarkable feature was the non-eroding of a bridge of bone extending from one anterior clinoid process to the other, forming the optic groove, and upon which rested the optic commissure. The lesser wing of

the sphenoid was not destroyed, saving the optic foramen and protecting the optic commissure and nerve from direct injury. The cribriform plate of the ethmoid, anteriorly, was not involved, and the roof and posteroexternal wall of the orbit were intact.

The pituitary gland was suspended in the midst of the broken-down, caseous tumor mass, but without apparent infiltration. The metastatic deposit had pressed upon and destroyed the third, fourth, the ophthalmic and superior maxillary branches of the fifth, and the sixth cranial nerves, explaining the sensory disturbances mentioned in the clinical history, as well as the ocular changes. The definite focalizing symptoms referable to this deposit were clearly evidenced at the autopsy, which at first led to the opinion that the intracranial growth was the primary trouble. The extensive area involved by the sarcomatous growth may be seen by referring to the accompaning illustrations. The character of the growth is that of a spindle-cell sarcoma.

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THE LYMPHATIC DRAINAGE OF THE FAUCIAL TONSILS.[1]

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That various micro-organisms may gain access to the general system through the tonsillar tissues of the throat has been so firmly established that it is not necessary again to demonstrate the absorbing properties of the tonsils and to point out the various infections which may enter through this portal. The fate of the micro-organisms which have entered the parenchyma of the tonsil is dependent upon two factors: first, the pathogenic potency of the germ itself, and, second, the vital resistance offered by the tissues to its invasion. If a non-pathogenic germ enters a crypt it is destroyed probably by the phagocytosis of outwandering polymorphonuclear leukocytes. On the other hand, if the vitality of the germ is sufficiently great, it lives and may penetrate through the epithelium into the interfollicular tissue.

It has been proven by several very thorough and capable investigators that foreign bodies in the crypts can pass through the epithelium into the interfollicular tissue. This absorption is probably dependent upon two factors: first, the action of the muscles of the throat during their various physiological movements, and, second, the presence of a lymph current in the tonsil. The palatoglossal muscle and the palatopharyngeal muscle during deglutition compress the faucial tonsil and force the centrally lying movable bodies toward the periphery. If the foreign body is at the bottom of the crypt it is pushed in an outward direction, and, finding no appreciable barrier in the differentiated epithelium, passes into the tonsil parenchyma. The second factor in the absorption of foreign bodies from the crypts is the constant production of lymphoid cells in the follicles. The interfollicular tissues of the tonsils consists of a fine connective-tissue reticulum interspersed with numerous lymphoid cells. These lymphoid cells may be looked upon as being in constant motion, passing from the follicles in the direction of least resistance. If the cellular metamorphosis of the epithelium has weakened this barrier so that the crypt is in the direction of least resistance, the lymphoid cells break through and pass into the crypt, but more commonly the lymphoid cells pass into the lymph spaces which terminate in the trabeculæ of the tonsil and which in their turn empty into the efferent lymphatics of the tonsil. Therefore, the production of lymphoid cells in the follicles causes what we

[1] Candidate's thesis. American Laryngological Association, June, 1905.

might term a lymphoid current, and this lymphoid current, except when there is an accidental rupture of the cryptal epithelium, is toward the connective-tissue trabeculæ of the tonsil and thence through the lymph radicles to the periphery. A foreign body having gained access to the interfollicular tissue must be influenced by this current. I understand thoroughly that in advancing this idea, I am open to criticism for theorizing about a scientific subject. It seems to me, however, that the above is a probable explanation of the mechanism of tonsillar absorption and helps to substantiate the theory that the faucial tonsil is more important as a source of infection than is the lymphoid tissue on the lateral folds and posterior pharyngeal wall of the pharynx. As regards the absorption of living virulent micro-organisms, it must be remembered that bacteria are not inert foreign bodies, but possess certain vital properties which influence their entrance into living tissue. When uninfluenced by extraneous forces a micro-organism invades living tissue by two methods: first, if it belongs to the motile class the individual bacterium by its own locomotion may carry itself to some more or less distant spot, and, second, micro-organisms may invade tissue by the very act of their growth. This last is the most important method by which the pathogenic germs gain entrance into their host.

It is practically impossible to establish with absolute certainty the relative importance of the different lymphatic masses in the throat as gates of infection. Circumstantial evidence, however, both clinical and experimental, tends to show that the larger the lymphatic mass and the deeper its crypts, the more readily do pathogenic germs pass through the tonsillar tissues to the tonsillar efferents.

The research outlined in this paper is a preliminary report of a series of investigations concerning the lymphatic anatomy and drainage of the tonsils and surrounding portions of the throat. My first investigations concern the faucial tonsil, because in the etiology of infections it is the most important tonsillar structure, and I have not been able to find any reliable published data concerning the glands that receive its efferent lymphatics.

The lymphatic glands of the neck constitute one of the most important chains of glands in the whole body and is probably the chain which is most frequently diseased. The infection which inoculates a lymph gland may possibly come through its efferent lymphatics by a retrograde thrombosis, but much more frequently it follows the current of the lymph stream and enters through the afferent vessels. In the large majority of cases the infection of the cervical lymphatics comes from the nose, mouth, or throat. As a general rule, the efferent lymphatic vessels from a given structure drain into that lymphatic gland which is nearest it, and, therefore, to understand the importance of the different structures of the nose, throat, and mouth as portals of infection it is essential to have clear

in our minds the topographical relations of the deep and superficial lymph glands.

The cervical glands of the neck are divided into two main groups, the superficial or collecting glands and the deep or terminal glands. The superficial are arranged as a sort of collar around the upper part of the neck, with a few irregular extensions. This pericervical glandular circle is composed of the following subgroups:

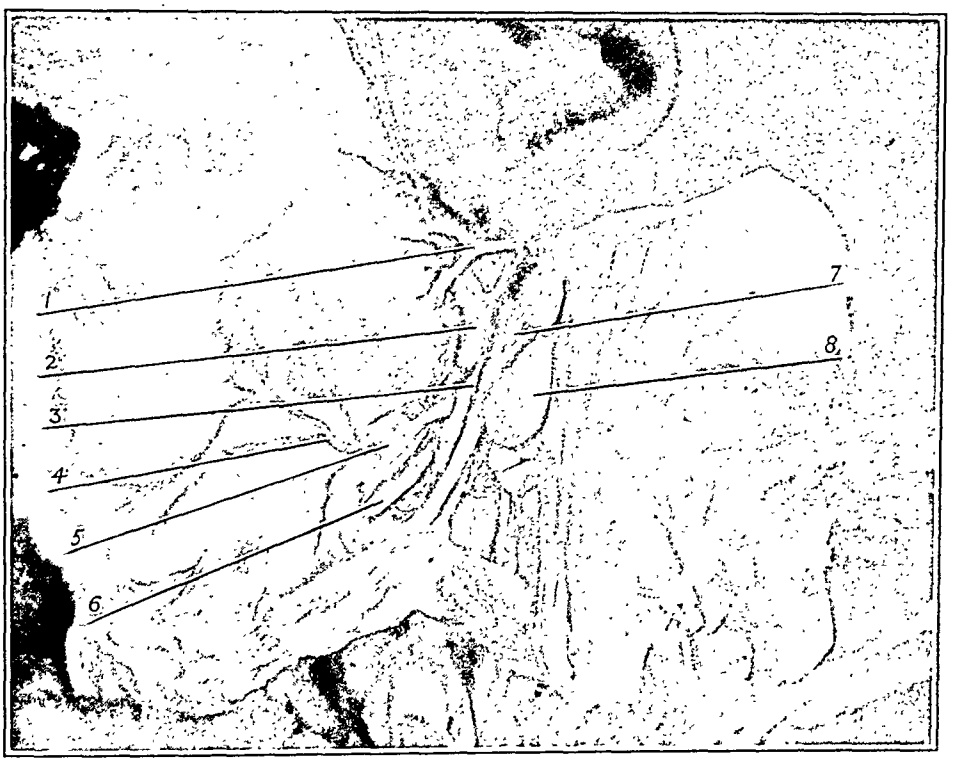
1. Suboccipital group and aberrant glands of the nape of the neck.
2. The mastoid group.
3. The parotid and subparotid groups.
4. The submaxillary group with the facial glands as an offshoot.
5. The submental glands.
6. The retropharangeal glands.

The terminal or deep glands of the neck form a perpendicular chain beneath the sternocleidomastoid muscle. This main set of glands is flanked by several secondary chains of less importance. The superficial glands constituting the pericervical collar receive their afferents from those portions of the external and internal head, which are in more or less close relation with the individual group. The efferents of all these collecting glands empty into the deep chain. The deep cervical lymphatics or the substernocleidomastoid glands constitute in appearance a continuous chain, but for convenience sake they may be divided into an external and internal group. The external glands are placed posteriorly and rest indiscriminately on the insertions of the splenius, levator anguli scapulæ, and scalene muscles, and they are usually continuous with the glands occupying the supraclavicular triangle. The internal glands of the substernocleidomastoid group may be termed the internal jugular chain, because they rest either directly on the internal jugular vein or are immediately adjacent to its external border. The glands of this subgroup are larger than the glands of the external, and some of them are fairly constant in their position. One or two large glands are constantly found beneath the posterior belly of the digastric, just above the spot where the thyrolingual facial vein opens into the internal jugular. A few glands are sometimes found between the internal jugular and the prevertebral muscles. These two main groups of deep glands, the external and internal chains, are united by a great many anastomoses. The external glands receive their afferents from the posterior part of the head and neck, while those of the internal group receive their afferents from the anterior portion of the neck and head, from the mouth, nose, pharynx, larynx, and thyroid body. While the majority of the afferents of the deep glands of the neck are the efferents from the superficial glands, quite a number of lymph vessels pass directly from the lymph radicles of the throat and nose to the deeper chain of glands. For instance, the majority of the lymph vessels from the vault of the pharynx

drain into the retropharyngeal glands, but some of them pass directly to the upper glands of the deep cervical chain without any interruption.

The method of injection employed in the present research was essentially that devised by Gerota. It consisted in rubbing up Berlin blue with pure spirits of turpentine until it assumed the consistency of a rather thin, syrupy liquid. A small quantity of ether is added and the mixture filtered through chamois skin. The coarser granules of the Berlin blue are held back in the filter while a clear, thin, blue liquid passes through as the filtrate. Gentle agitation of the

FIG. 1.



Dissection of the neck of a child, showing an enlarged tonsillar lymph gland and its relation to the digastric and stylohyoid muscles: 1, facial nerve; 2, external carotid artery; 3, stylohyoid muscle; 4, facial artery; 5, submaxillary lymph gland; 6, hypoglossal nerve; 7, posterior belly of the digastric muscle; 8, tonsillar lymph gland.

fluid while it is filtering ensures a better mixing of the turpentine, ether, and Berlin blue, consequently a deeper color to the filtrate. The injecting apparatus consisted of a fine glass needle drawn out of a glass tube and attached by a piece of rubber tubing to the nozzle of an easy-working syringe holding about 10 c.c.

The glass needle was inserted beneath the mucous membrane covering the tonsil and held very carefully in its place, while an assistant made gentle but gradually increasing pressure, according to the ease with which the fluid entered the tonsil. The amount injected was regulated somewhat by the amount of resistance felt in

the syringe and also by the distention of the tonsillar tissues. If the injection was properly made the tonsil gradually swelled out, becoming two or three times its normal size and of a deep-blue color. The blue fluid in such injections must not be forced in hurriedly, as a certain amount of time is necessary for it to find its way into the lymph spaces and thence through the lymph radicles to the lymph vessels. In injecting the tonsil, it frequently happens that the fluid returns through the crypts. In the case of this regurgitation being so free that internal pressure cannot be exerted, the point of

FIG. 2



Dissection of the neck of an adult, showing enlarged internal jugular lymph glands: 1, parotid gland; 2, masseter muscle; 3, facial nerve; 4, facial artery; 5, submaxillary lymph gland; 6, submaxillary salivary gland; 7, thyrofacial lingual vein; 8, internal jugular vein; 9, brachial plexus of nerves; 10, superficial cervical plexus; 11, spinal accessory nerve; 12, internal jugular lymph glands.

the needle is to be changed until a position is found from which the injected material does not so readily gain access to the crypts. After the injection has been made the excess of material should be washed from the surface by a stream of cold water. Gentle massage of the tonsil with the finger tends to force the injected fluid farther into the tissue. It is best to leave the fresh material in plain water for several hours before fixing it in 10 per cent. formalin. After fixing the specimen the dissections may be made and the injected gland sought for, special care being exercised to avoid wounding any of the injected lymph vessels.

Eight injections were made, some of them resulting in failure, but the majority showing distinctly that the tonsillar lymph follows a fairly constant route. The direction of this drainage as established by these injections is as follows:

The lymph vessels pass from the external portion of the tonsil through the peritonsillar connective tissue, the pharyngeal aponeurosis, and the superior constrictor of the pharynx, and, as one or two or more fine small vessels run obliquely in a downward, posterior, and outward course, passing below the facial artery. Bending more posteriorly the lymph vessels next run between the internal

FIG. 3



Superficial dissection of Specimen No. VI., showing enlarged tonsillar lymph gland projecting just beyond the edge of the sternocleidomastoid muscle: 1, external jugular lymph glands; 2, great auricular nerve; 3, tonsillar lymph gland; 4, external jugular vein; 5, external glands of the substernomastoid group; 6, submaxillary salivary gland; 7, submaxillary lymph glands; 8, facial artery.

jugular vein and the stylohyoid muscle, reaching finally the superior surface of an enlarged lymph gland, placed just beneath the anterior border of the sternocleidomastoid muscle, where it is crossed by the posterior belly of the digastric muscle. The efferent vessels from this gland are generally two or three in number, and pass into the neighboring glands of the internal jugular group. Further anastomoses which connect the lower glands of the internal jugular group with those receiving the tonsillar drainage form a complete lymph channel, through which the tonsillar lymph finally empties into the jugular lymph trunk.

In none of the preparations did the injected fluid enter into superficial glands, except in one case, where there was an aberrant gland lying on the facial artery near its origin. This gland seemed to be simply an interrupting nodule in the course of the tonsillar efferents. The statement in some text-books on anatomy that the tonsil drains into the posterior gland of the submaxillary group would seem to be absolutely erroneous and very misleading.

Clinically, the gland which becomes enlarged during tonsillar infection appears to be superficial and possibly it is this appearance that has led to the belief that the posterior gland of the submaxil-

FIG. 4

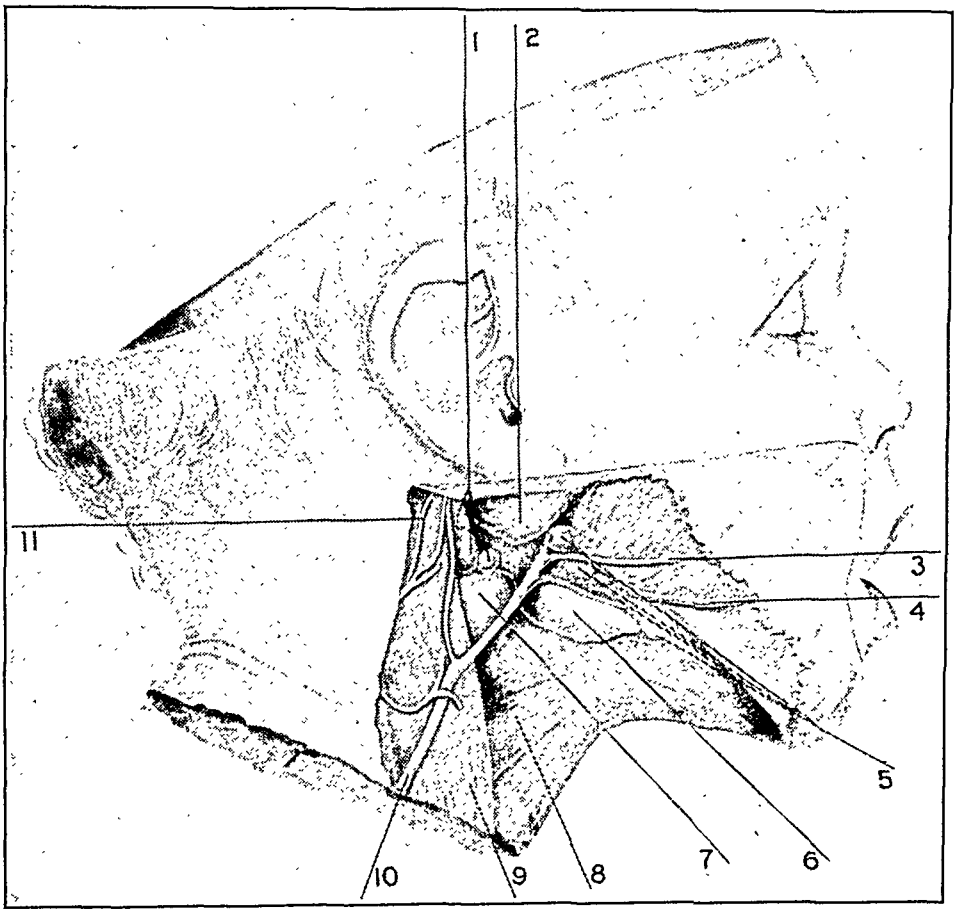


Deep dissection of Specimen No. VI., showing the tonsillar lymph gland and its relation to the substernomastoid group: 1, reflected sternocleidomastoid muscle; 2, tonsillar lymph gland; 3, spinal accessory nerve; 4, branch of cervical plexus; 5, internal jugular vein; 6, omohyoid muscle.

lary group is infected through the tonsils. The tonsillar gland, if I may be pardoned for using the term, is placed external and slightly anterior to the internal jugular vein and is embedded in loose areolar tissue containing more or less fat. Consequently, enlargement of this gland means its dislocation outward and forward, and especially so if the other glands of the internal jugular group become subsequently enlarged; also the action of the substernocleidomastoid muscle would tend to force it anteriorly. If an enlarged gland found at the angle of the jaw belongs to the deep substernocleidomastoid group, it may be pushed back under the muscle of the same

name. In cases of enlargement of the submaxillary glands such as result from infection from carious teeth, the glands are found along the edge of the jaw and cannot be displaced backward and only slightly downward. In the description of the anatomy of the cervical lymphatics previously given it was stated that where the posterior belly of the digastric muscle crossed the sternocleidomastoid there were constantly found one or two large glands. In a child one week

FIG. 5

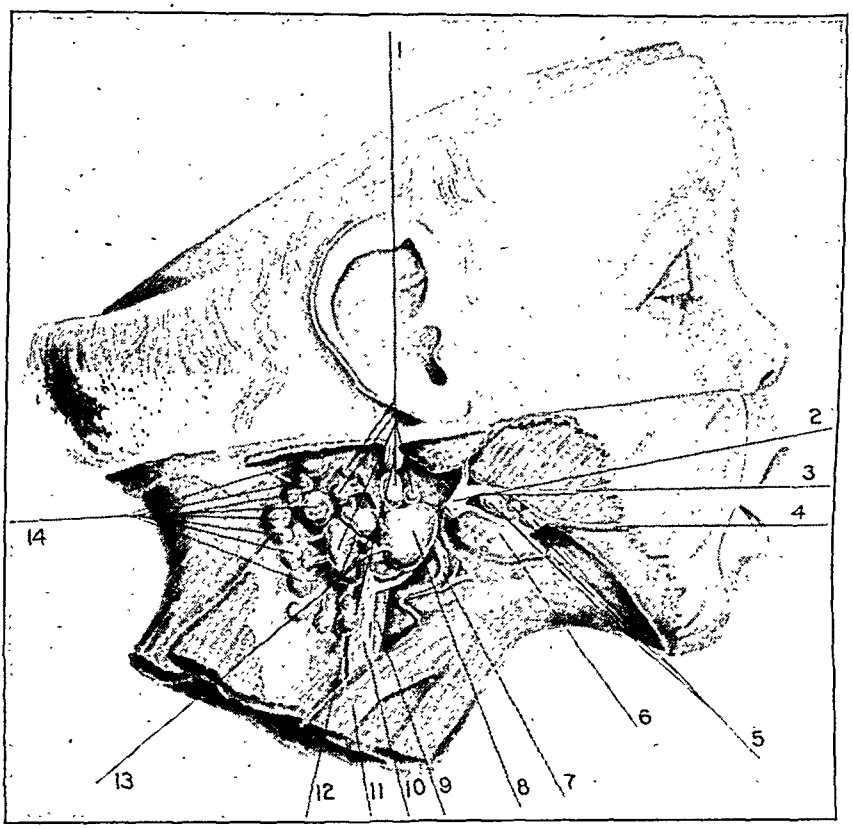


Superficial dissection of Specimen No. II., showing portion of tonsillar lymph gland and its relation to the sternocleidomastoid muscle and the external jugular vein: 1, lymph glands of the internal jugular group; 2, parotid gland; 3, facial vein; 4, facial artery; 5, submaxillary lymph glands; 6, submaxillary salivary gland; 7, tonsillar lymph gland; 8, omohyoid muscle; 9, sternocleidomastoid muscle; 10, external jugular vein; 11, great auricular nerve.

old in which I was able to trace the course of the tonsillar efferent vessels this gland was very little larger than many more of the external jugular group. In children who have reached six or more months of age this gland was generally twice or three or four times larger than any other, and in every injection of the tonsil it was this enlarged gland that primarily received the blue injecting fluid. Does it not seem possible that this enlargement consequent to birth may be due to the absorption of toxins through the faucial tonsils?

I have chosen to call the lymphatic gland which receives the efferents of the tonsil the *tonsillar lymph gland.* This term is not sufficiently comprehensive, because most probably other lymph vessels from the throat, besides those from the tonsil, drain into this gland, but the most important infections of the cervical lymphatics originate through the tonsil, and because of the importance of the

FIG. 6



Deep dissection of Specimen No. II., showing position of tonsillar lymph gland and its connection with other glands of the substernomastoid group: 1, internal substernomastoid lymph glands; 2, external jugular vein; 3, facial vein; 4, facial artery; 5, submaxillary lymph glands; 6, submaxillary salivary gland; 7, superior thyroid artery; 8, tonsillar lymph gland, 9, common carotid artery; 10, internal jugular vein; 11, omohyoid muscle; 12, jugular lymph trunk; 13, efferent vessels of tonsillar lymph gland; 14, external substernomastoid lymph glands.

tonsil in the origin of infections, I feel that the term tonsillar is a most appropriate one. Certainly, it is convenient to be able to easily designate so important and constant a lymphatic gland.

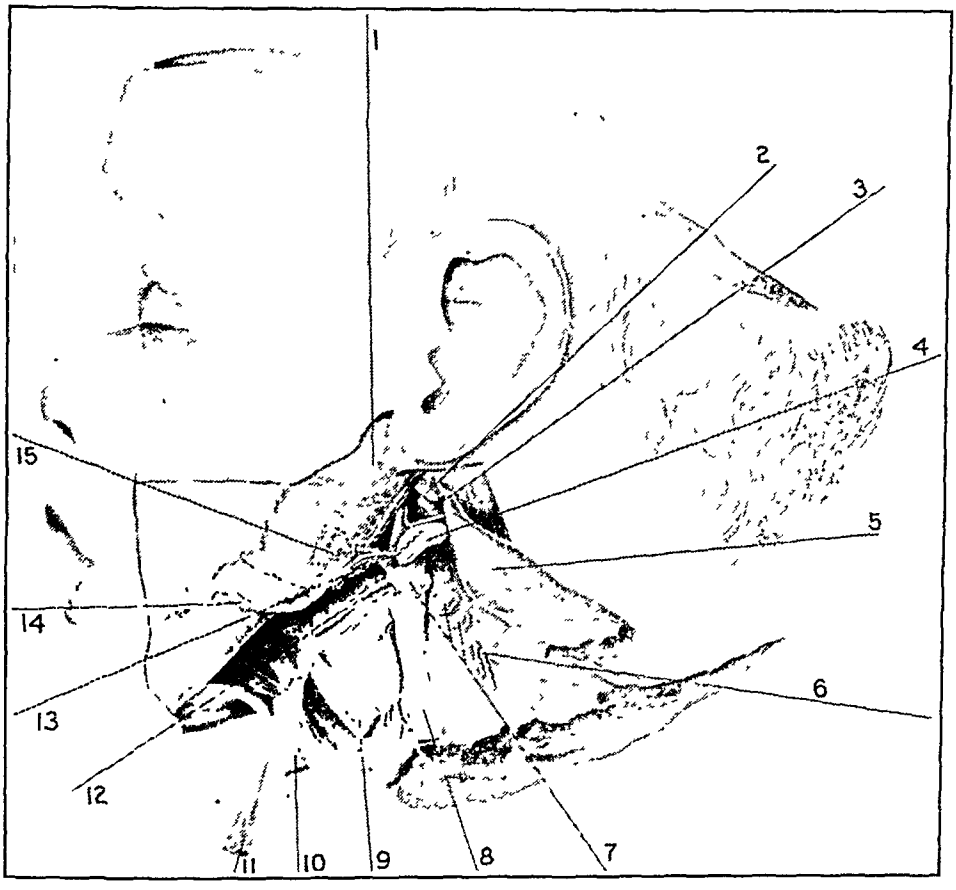
Following are the descriptions of the individual injections of the tonsils. The technique was practically the same in all of them.

SPECIMEN I.—Child about six months old. The injection in this case failed probably because there was some obstruction in the

injecting needle. Dissection of the neck showed hypertrophy of the tonsillar lymph glands.

SPECIMEN II.—Child about six months old. The injection in this case was very successful. The tonsil itself became enlarged and there was a slight overflow of the fluid toward the faucial pillars, the blue color, however, was limited to the fauces. A few injected lymph vessels were seen running posteriorly into the sinus pyriformis

FIG. 7



Deep dissection of Specimen No. IV., showing the efferent lymph vessels of the faucial tonsil entering the tonsillar lymph gland. The posterior belly of the digastric muscle and the whole of the stylohyoid muscle have been reflected downward: 1, parotid gland; 2, stump of the stylohyoid muscle; 3, stump of the posterior belly of the digastric muscle; 4, tonsillar lymph gland; 5, reflected sternocleidomastoid muscle; 6, brachial plexus of nerves; 7, substernomastoid lymph glands; 8, internal jugular vein; 9, omohyoid muscle; 10, stylohyoid muscle; 11, posterior belly of the digastric muscle; 12, hypoglossal nerve; 13, efferent lymph vessel of the faucial muscle; 14, facial artery; 15, position of faucial tonsil.

and anteriorly toward the base of the tongue. The dissection of the neck showed that the blue fluid had entered a slightly enlarged lymph gland situated just below the point where the anterior border of the sternocleidomastoid muscle is crossed by the posterior belly of the digastric. The gland was superficial to the digastric muscle just above the facial vein, where it joins the external jugular. A

deeper dissection demonstrated that the injected gland belonged to the anterior group of the upper deep cervical lymph glands and had three efferent vessels, which also contained the blue injecting fluid. The connecting lymph vessels between this gland and the jugular trunk were dissected out. The most direct route showed that the lymph current from the tonsil, in order to enter the jugular trunk, had to pass through two or more glands after leaving the tonsillar lymph glands. In this specimen, however, I was unable to find the efferents running from the tonsil to the tonsillar lymph gland. Figs. 5 and 6 were made from this specimen.

SPECIMEN III.—Child one week old. Only a small quantity of fluid was injected into the tonsil. The dissection showed a slight injection of the glands situated just below the posterior belly of the digastric.

SPECIMEN IV.—Child one week old. A rather full injection of the tonsils was made and there was some infiltration of the surrounding mucosa. This injection was very instructive, as by careful dissection lymphatic vessels could be traced running from the infiltrated tonsil directly to the gland situated just below the posterior belly of the digastric beneath the anterior border of the sternocleidomastoid muscle. This lymph gland was considerably injected, but the importance of the specimen was in the demonstration of the efferent lymphatics of the tonsil. The course which the lymphatic vessels took was as follows: Leaving the tonsillar mass and running posteriorly and inferiorly, they pass slightly outward beneath the facial artery, running along the upper border of the lingual vein, then turning directly posterior, they pass between the stylohyoid muscle and the internal jugular vein, reaching the upper surface of the tonsillar lymph gland. Fig. 7 was made from this specimen.

SPECIMEN V.—Child about three months old. Too much fluid was injected in this specimen, and, although the tonsillar lymphatic gland and its efferents contained the blue solution, the extravasation through the connective tissue of the neck was so great as to make it impossible to recognize the topography.

SPECIMEN VI.—Child about three months old. This injection was a most successful one. Quite a large amount of fluid was injected in this case without any extravasation. In the throat the fluid was limited to the confines of the tonsil and the dissection showed that it had followed the lymph vessels through six or more of the upper deep cervical glands. The efferents from the tonsil could be traced passing in a direction very similar to that described under Specimen IV. There were two or three lymph vessels which ran closely together in a more or less parallel course. At first they passed obliquely downward, posteriorly, and outward until they reached the facial artery. Running beneath this and internal to the stylohyoid muscle and the posterior belly of the digastric muscle, they entered the internal side of the tonsillar lymph gland.

In closing, I desire to express my thanks to Dr. James M. Stotsenburg and especially to Dr. Harold D. Senior for their skilful aid in making the injections.

CONCERNING THE OBSCURITY OF DIAGNOSIS SOMETIMES ATTENDING STONE IN THE KIDNEY; WITH REPORT OF CASES.[1]

By D. D. Stewart, M.D.,[2]
of Philadelphia.

The presence of stone in the kidney is commonly apt to be regarded as fairly easy of determination when it exists in other than a quiescent state, and is suggested by the usual direct symptoms—the occurrence of loin pain, paroxysmal or continuous, with exacerbations aggravated by exertion, shooting into the groin, testicle, and thigh, and with the presence of blood in the urine. It is, however, well understood that various other morbid conditions of the kidney—the presence of a neoplasm; slight or, again, easily ascertainable displacement of the organ causing torsion of the ureter; intermittent hydronephrosis, so produced or induced by a ureteritis; so-called "nephralgia" and the like—all simulate stone, and may cause its presence to be suspected when non-existing. Eshner[3] recently in reporting a case, the symptoms of which simulated stone, has painstakingly collected from the medical literature a rather extensive series of cases in which, with unilateral renal hæmaturia as the chief objective symptoms, stone, although suspected in a number, was later regarded as present in none.[4]

With symptoms suggesting stone, the diagnosis is now materially helped by the Roentgen ray, and yet both its positive and its negative finding may be misleading and sometimes mischievous; not least as concerns the latter is the fact that with too blind dependence on its reading an abnormality other than stone, requiring surgical interference, existing, may escape consideration, and the ailment be regarded as a "nephralgia," although actually due to a condition only amenable to operation, such, for instance, as torsion of the ureter, the result of a slight or greater degree of mobility of the kidney, to a ureteritis, thus or otherwise produced, or to a kidney,

[1] Read before the Twentieth Annual Meeting of the Association of American Physicians at Washington, May, 1905.

[2] It is the sad duty of the Editor to note that this is a posthumous article. Dr. Stewart died on June 13, 1905.

[3] Unilateral Renal Hæmaturia, The American Journal of the Medical Sciences, April, 1903.

[4] Although it should be said that the technique of the operative procedure for the recognition of stone was not always above reproach.

the functions of which are hampered by adhesions, as in one of the cases I report.[1]

It is said that renal calculus may rarely exist for a long time symptomless. The condition of absolutely quiescent renal calculus the writer doubts, and regards such cases as those not carefully studied as concerns past history and the urinary condition.

Presumed affections of other organs may be masqueraded by renal calculus, the symptoms of stone being so referred; and contrariwise, gastric ulcer, gallstones, abdominal tumors, and affections of the bladder and ovaries may simulate calculus. It is only recently that the frequency of the occurrence of loose kidney causing symptoms—*i. e.*, intermittent torsion of the ureter, with moderate hydronephrosis—has received the attention it should as a cause of loin and abdominal pain. In this connection the writer has in mind a case seen a few years since with Dr. Lewis Brinton, in which, because of recurring attacks of severe pain in the region of the loin and groin, the patient, a young woman, before she came into Dr. Brinton's hands had been operated on for suspected appendicitis, and the appendix, presenting a healthy appearance, was not removed. The ovary, although equally healthy, was, since no other source for the ailment could be imagined. On recovery from the cœliotomy the pain recurred as before. The surgeon now by the process of exclusion regarded his earlier diagnosis as correct and urged a second

In the hands of a few experts the *x*-ray has proved of much service in the diagnosis of renal and ureteral stone, but even with these its negative finding cannot always be depended upon. The point of vital importance in its use seems to lie in the axiom which I think was first propounded by Leonard, that "where shadows of tissue less dense than the least dense calculus are shown in the skiagraph no calculus can escape detection." In other words, tissue differentiation must be apparent." Cole's rule (April 22, 1905, p. 761, cited by Brewer, Medical News) is an amplification of this, "that a diagnosis is not positive unless the outline of the psoas and the transverse processes of the lumbar vertebræ are distinctly visible in the radiogram, and, further, that the edge of the shadow of supposed stone be closely and clearly defined." Unfortunately there are few, even among those regarding themselves as competent radiographers who, if alive to this vital rule, exemplify it in their work, whence arise errors.

It may be all very well to dilate on clear definition of stone and differentiation of the psoas, and to remark, as does a recent writer (Dr. Bevan, Medical News, April 30, 1905), that the *x*-ray has absolutely no fallacies, but it remains true, as Dr. Bevan also states, that "most of the work done with the *x*-ray in kidney stone has been poor work." Skiagraphers especially skilled in this field are not numerous, and even these are unprepared to admit absence of error. Leonard's statistics, which are remarkable when compared with the work generally done, show a failure to find but four calculi in 103 cases, out of a total of 331 suspects examined. His total errors in this number to the present are ten. One of the cases in which Dr. Leonard failed to discover a stone is the first of these related in this paper. In this case the stone was composed largely of calcium oxalate and was of fair size, and the anteroposterior diameter of the patient was not great. Two examinations were made at first by Dr. Sweet, and again two at my request by Dr. Leonard. The latter was positive no stone existed, until it later was removed by Dr. Keen. This, however, was over five years ago, but at that time Dr. Leonard had already realized the importance of differentiation of shadows less dense than the least dense stone; improvement in technique has doubtless occurred since. It is often difficult to interpret the *x*-ray plates in cases of suspected calculus, and not infrequently positive diagnoses err. Thus, periureteral bodies of different origin, such as a calcified tip of an appendix epiploica, have positively indicated ureteral stone (Brewer, Medical News, April 22, 1905, p. 761), and Caldwell also points out that renal and ureteral stones have been masqueraded by pseudoshadows in the radiograms of uncertain origin, and that these have deceived experienced *x*-ray operators.

operation for appendectomy. The patient was now brought to me by Dr. Brinton; a loose, large, and painful right kidney was demonstrated. The symptoms were clearly those of slight intermittent hydronephrosis. An immediate nephropexy was advised. This was performed by Dr. W. E. Ashton. The patient was immediately relieved of, and has since remained free from all discomfort.

It has not been altogether unusual in my experience to see cases with affection of one kidney variously diagnosed as appendicitis and as gastrointestinal disease. I recall a Polyclinic Hospital case, seen with Dr. T. S. K. Morton a few years ago, in which appendicitis had been suspected, but in which there was a frank history obtainable of intermittent hydronephrosis, and in which the ureter could be palpated as a thickened cord—here an obstructive ureteritis had originated the symptoms, and the thickened ureter suggested an elongated diseased appendix. I have in mind another case sent me by a prominent surgeon as one of dyspepsia. The patient had various abdominal symptoms and had been under casual treatment by the surgeon and his assistant for months as gastric catarrh. The abdomen, in the hurry of work, had not been examined. The usual routine examination at the first visit disclosed a large and very loose right kidney, and a urine examination showed a pyelitis. Recovery followed a nephropexy. There are cases other than stone in the kidney in which calculus was not suspected, but in which a distinct kidney abnormality often associated with stone had been referred to another organ, and a too casual examination had at first caused error in diagnosis. They are referred to as in line with remarks that follow as to diagnosis of some special aspects of renal calculus and of kidney conditions simulating stone.

The purpose of this paper is not to discuss the general subject of diagnosis of renal calculus, but to report a small group of cases—cases with calculus and without it, though simulating stone, which, different in character, present features of such interest as to render their detail worth while from the standpoint of diagnosis.

Two of these were cases of unsuspected calculus, and one at least "masked" calculus, in neither of which had the diagnosis been made until seen by the writer. These, as well as Case III., the exact nature of which is still in doubt, were cases in which the symptoms had not been regarded as suggestive of an affection of the kidney. In the first of these (renal stone) there were absolutely no direct symptoms. In the others, as in Case IV., they were only developed by careful inquiry, and the probable diagnosis in each could only be made by exclusion and by a study of the urine. One of these cases of stone had been in the hands of a surgeon of wide experience for months, and was treated as a case of dyspepsia before he was referred to the writer.

In another case which, with symptoms also not directly referable to the kidney, had been previously diagnosed as one of neurasthenic

gastralgia, I had suspected stone in the kidney, but an exploration of that organ by Dr. Keen indicated dense adhesions and ureteritis as the cause of the pain. In another, the history of which precedes the foregoing, from the first stone had been diagnosed frank renal colic with recurring hæmaturia occurring, a strictured ureter without stone was found. In another with symptoms strongly suggestive of stone, in which explorations of the kidney had been performed by Dr. Halsted, the presence of calculus was still suspected by the latter and similarly by myself; later developments demonstrated the case to be one of pure hysteria, the renal colic apparently having been a mere expression of this condition. Doubt was felt for a long time as to the correctness of diagnosis, because of the deception practised by the patient. It is especially desired to emphasize the fact that renal calculus and allied conditions of the kidney will sometimes produce symptoms which express themselves chiefly or alone as a disturbance in the intestinal tract and that such cases may be overlooked unless repeated examination of the urine be made. Attention must be called particularly to the first case to be related, in which the ailment would have been regarded as a colitis but for the result of a study of the urine; also to that of the case which follows this, in which the suspicion of kidney stone, when the patient was first seen, was converted into a strong probability, on an examination, of a specimen of urine voided during the initial visit, showing the presence of considerable (microscopic) blood. In the case of C. M., referred to me as one of presumed bowel trouble, it was only after several visits that a specimen of urine was obtained; then the presence microscopically of considerable erythrocytes and their persistence in each specimen examined subsequently, with absence of urethral or bladder disease, caused me to suspect stone. Here so unusual was the absence of direct symptoms that operative interference was refused by a surgeon, to whose hospital service I at first sent him. The history of the case is as follows:

CASE I.—C. M., aged twenty-three years; weaver. Referred to me by Dr. R. H. McCamy on October 15, 1898. Symptoms of bowel disorder. The history then elicited was: He had developed chronic diarrhœa when about seven years of age, which had lasted some six years. There had been a good deal of abdominal pain with this, from which afterward he had never been entirely free. When he consulted me there were soreness and pain extending across the abdomen in the direction of the transverse colon, to the extent of several inches, and thence downward for a short distance following in the line of the descending colon and sigmoid. Here at one point (see diagram), two and one-half inches below and one and one-half inches to the left of the umbilicus, there was marked tenderness on pressure, and about this area colicky pains would occur. With this pain, and apparently aggravating it, there was a good deal of flatulent distention of the colon. Physical examination disclosed a

myasthenic, dropped stomach, in which a very audible splash sound was present five hours after food and drink. Marked distention of the bowel and rigidity of the abdominal wall prevented the left kidney from being readily palpable. On two occasions its lower pole was detected, but sensitiveness could never be elicited by manipulation of the left loin.

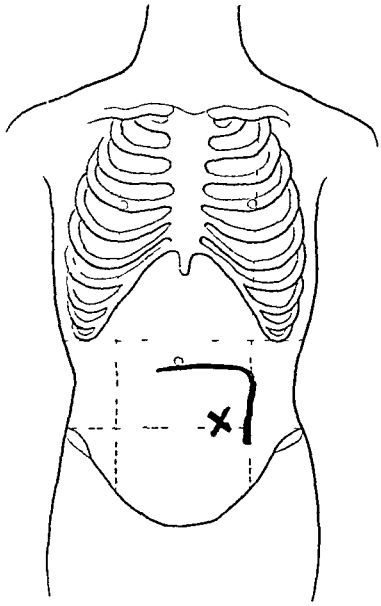
The symptoms suggested a colitis, and only the usual routine examination of the urine prevented this mistake being made. The urine was clear; of amber color; specific gravity 1020; deposited but a light flocculent sediment after some hours' standing. A fair trace of albumin (heat and acetic acid) was present, and microscopically an uncentrifuged specimen showed many leukocytes and a few red blood corpuscles, with a moderate number of hyaline casts and cylindroids.

Further questioning now elicited the history of a strained back, through very heavy lifting, four years before. About a year following there was a good deal of pain in the dorsal region and in the left side. This afterward disappeared, and the only pain he had had lately was that before mentioned in the region of the transverse and descending colon. There was no history of gonorrhœa or of past or present bladder trouble. The patient was now seen a half-dozen times at intervals of one to two weeks. During this time careful study of the urine was made both by Dr. Ghriskey and myself. There were always present a fair trace of albumin, many erythrocytes and pus corpuscles, hyaline casts, cylindroids, and streptococci. Occasionally cells, presumably from the renal pelvis and from the ureter, were found. Coincident with marked aggravation in attacks of abdominal pain, toward the end of December, red blood corpuscles and pus cells were present in the urine in much increased number, but the blood or pus never was evident macroscopically. The urine was always of acid reaction, of normal color, specific gravity 1017 to 1020, 1000 c.c. in twenty-four hours, and never contained more than traces of free uric acid crystals. Calcium oxalate crystals were never seen. Tubercle bacilli were examined for on a number of occasions resultlessly. Marked variation in the amount of urine voided from day to day was carefully watched for for some time, but did not occur. The patient had never had urethral or bladder disease.

The pain had become more paroxysmal, especially when about and at work, yet would often appear before rising in the morning, and at times was very severe; there never occurred any indication of renal colic, or of radiation of pain into bladder, testicle, or thigh. The pain followed the path before described from the umbilicus, across and thence downward for two to three inches in the line of the descending colon, and was at first thought to suggest a colitis. It was associated with flatulent distention of the bowels and was relieved by the expulsion of gas.

I regarded the case as either slight urethral stricture from a preceding ureteritis, the result of past injury to the kidney, the result of traumatism (strain), or one of renal calculus. The man was a poor weaver, with a family dependent upon him, and was most adverse to abandoning work for the purpose of exploratory operation. His consent was finally obtained in January, three months after he first came under observation, at a time when he was temporarily out of employment. I then referred him, for exploration of the kidney, to the surgical ward in one of our hospitals. Unfortunately, the surgeon on duty, after an examination of the case, and detaining him

FIG 1



The cross marks the situation of crampy pain and of sensitiveness, the right angle the situation of persistent soreness, suggesting colitis.

for some little time in the wards for observation, refused operative interference, not regarding the symptoms as suggestive of a kidney or urethral ailment. This rendered the patient thoroughly averse to surgical interference, and it was not until some months later, when I began my service in the Episcopal Hospital, that I could do anything further with him. I then had him admitted, for exploration of the kidney, to Dr. T. R. Neilson's ward. A skiagraph taken by Mr. Riegel indicated the presence of a rather large calculus in the left kidney, which the operation by Drs. Neilson and H. C. Deaver disclosed.

The stone, of triangular shape, lay in the pelvis of the kidney; one end, shaped like a pig's snout, engaged the mouth of the

ureter. The kidney was somewhat enlarged and placed a trifle lower than the normal. The stone was of good size when freshly extracted, weighing eighty grains. It was made up in very nearly equal amounts of uric acid and of calcium oxalate.

The accompanying sketch, made by my friend Dr. J. Madison Taylor, shows the exact size of the stone. For a description and very complete analysis of the stone I am indebted to Prof. H. F. Harris, formerly of the Jefferson Medical College. In concluding his report Dr. Harris remarks:

"From the various reactions given by the substance composing this calculus it appears certain that the nucleus is made up in very nearly equal amounts of uric acid and calcium oxalate, and that the outer portions are composed almost entirely of calcium oxalate, with a small amount of organic coloring matter and probably slight traces of other organic substances."

FIG. 2







Exact size of stone.

It is of interest that the following case of stone in the kidney had, prior to coming under observation, been treated for some months as one of dyspepsia by a surgeon of recognized skill. In the rush of a busy practice time had not been taken for thorough examination, and the presence of intestinal indigestion with marked aggravation of symptoms through dietetic errors misled.

CASE II.—W. H. K., aged thirty-six years; bookkeeper. Referred to me on December 1, 1899, by Dr. H. F. Hansell. The patient had had for over eighteen months pain in the abdomen, which had been diagnosed and treated by a half-dozen general practitioners and lately by a surgeon as gastrointestinal disorder. The pain was more or less continuous and of moderate severity, but with paroxysmal aggravation so severe as to "double him up." These paroxysms were usually of very sudden onset, striking him, as he described it, "like a shot." He would be practically free from pain, and then,

without physical effort on his part, pain would appear, and become of great severity. Pressure applied to the loin and abdomen by the hand commonly greatly relieved the pain. Such pressure he applied when walking about, and frequently at night on retiring. For a long time he had been unable to lie on the right side, because of a paroxysm being thus immediately induced. He could lie with moderate ease on the back. Walking about often relieved the lighter paroxysm, and pressure by the hand sunk into the loin and upward against the ribs would also give him relief. His digestion had not been good for a long time, and a slight dietetic error would invariably aggravate the pain. There was a great deal of bowel flatulency, but passage of gas was difficult. After the expulsion of flatus the pain would commonly be lessened. The pain never radiated toward the groin or into the loin.

Eating or drinking anything cold tended to induce a seizure. Although of fairly robust appearance, he had lost twenty-five pounds in the past year. The skin was of an unwholesome, bronze hue. His appetite was slight, and the digestion greatly impaired since the ailment first appeared.

Blood had never been noted in the urine by the patient, and he had at no time remarked frequency of urination or symptoms suggesting urethral obstruction. His symptoms, as narrated, were to me so strongly suggestive of renal calculus that I told him, before I examined him, that I thought this the probable diagnosis. Examination disclosed decided tenderness in the left loin, more marked posteriorly and anteriorly in the situation noted in the diagram. Along the middle part of the course of the ureter, in the region of the sigmoid flexure, palpation was very painful. The left kidney was deeply and so highly placed that on the fullest expiration, with the abdomen relaxed, its lower pole could only be made out by bimanual manipulation.

The freshly voided urine, examined at the time of his first visit, showed an abundance of red blood corpuscles, singly and in large groups, and a small amount of free uric acid crystals. The specimen was of high acidity. There was a decided trace of albumin. The result of a number of examinations subsequently made both by Dr. Ghriskey and myself before operation were similar to the above.

The *x*-ray examinations were made at my request by Dr. Sweet at the Jefferson Medical College, and two by Dr. Leonard at the University. As a result, both gentlemen gave the positive opinion that no stone existed. I referred the patient to Dr. Keen for exploratory operation, suggesting that the diagnosis lay between stone in the kidney and strictured ureter, renal calculus being the more probable, despite the lack of *x*-ray confirmation.

Operation by Dr. W. W. Keen on January 6th. The kidney was very firmly adherent and placed somewhat higher than usual. It

was enlarged and deeply congested. A stone was removed from the pelvis. It was 12x10x5 mm. in measurement. An analysis made by Dr. W. M. L. Coplin showed that it was composed almost solely of calcium oxalate.[1]

FIG. 3

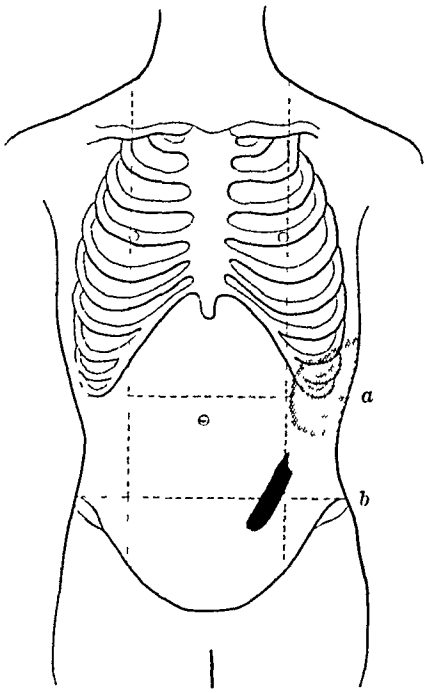


FIG. 4

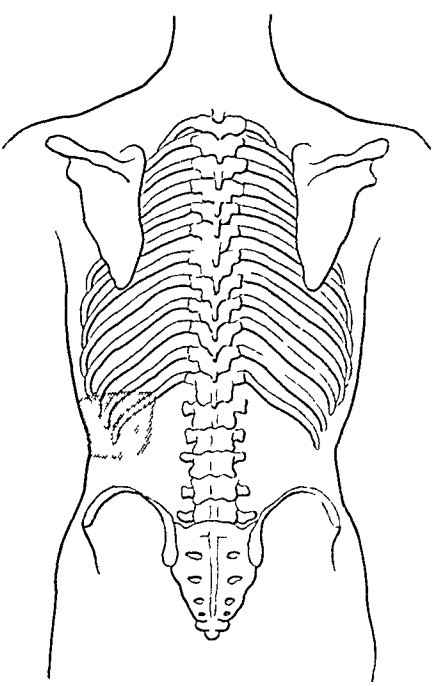
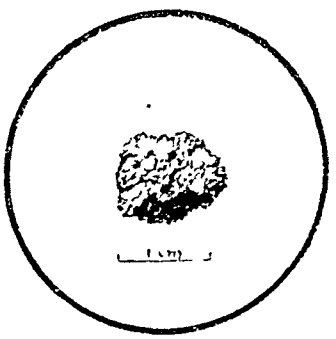


FIG. 3.—Anterior area of pain : *a*, area of pain ; *b*, very sensitive to pressure. Shooting pains in this area, but never to groin or kidney.
FIG. 4.—Posterior area of pain.

FIG. 5



The patient made a rapid and uninterrupted recovery, even while abed, two weeks after the operation, taking and digesting with ease food that previously he had not been able to eat for nearly two years.

[1] I am indebted to Prof Coplin for the accompanying photograph and for a very complete report of his analysis of the stone.

A case strongly suggesting the preceding one is now under observation, and is worthy of brief narration:

CASE III.—H. M., aged fifty-five years; merchant; first seen March 22, 1905. Consulted me on account of "indigestion" and "uric acid in the system." For two or more years past he has had attacks, usually at night, of severe pain in the lower part of the abdomen, limited chiefly to the left side. The pain is associated with marked flatulent distention of the abdomen, "rumbling and rolling in bowels," and he thinks is so initiated. Expulsion of gas by bowel relieves the pain, but he is generally unable during an attack to pass flatus. Although not constipated, purging with salts assuages the pain. Rising from bed and walking about enables him to better expel flatus and thus tends to assuage the pain, but if the latter is severe a chill is apt to occur on getting up. There is only occasionally a tendency to frequent urination during the attacks. No alteration in color of urine observed and no gravel passed. No nausea or vomiting during a seizure. Appetite good; careless as to diet; rapid eater; teeth in good condition. No symptoms of stomach disorder. Much confined in-doors. Five to seven cigars daily; no alcohol; no lues. La grippe only ailment has ever had. No loss of weight.

Physical Examination. Color of skin normal. Rather high blood pressure; radials trifle thickened; no cardiac hypertrophy or valvular lesion; accentuated second sound at apex.

Abdomen much distended with gas, chiefly intestines; stomach undilated; umbilical hernia. Marked sensitive point always present two inches below and two and one-half inches to the left of the umbilicus—in the situation noted in the case of C. M. No sensitiveness or pain elsewhere in first examination. Examination of stomach contents showed normal secretory activity; delayed motility. Urine voided in laboratory: clear, light amber; acid; specific gravity 1011; albumin plus normal trace, microscope; no centrifuge used; cylindroids; uric acid crystals; leukocytes; few red blood corpuscles.

Provisional Diagnosis. Arteriosclerosis; abdominal pain on expression of the arteriosclerosis, or due to intestinal indigestion.

TREATMENT. Diet regulated; fresh air; no tobacco; blue-mass, followed by Glauber salt; morning dose of Carlsbad subsequently; tincture nux vomica and nitroglycerin. At bedtime betanaphthol, 5 grains in tincture ginger 1 drachm.

No improvement on the above. Pain occurred as before; stone in the kidney suspected. Naphthol discontinued; daily examination of urine made; all specimens (centrifuged) show red blood corpuscles, singly and in groups, with a fair number (about one-third in proportion to red blood corpuscles) of leukocytes; hyaline casts in small number in most specimens; polyhedral cells, presumably from tubules, similar to those making up two epithelial casts seen; few caudate cells, presumably from renal pelvis; uric acid crystals.

Severe attack of pain in left loin night of April 4th, with tendency to shoot toward pubes and into the inner side of thigh. Sensitiveness in the left loin laterally and posteriorly the morning after, and in the abdomen to the left and below the umbilicus. Urine voided (11 A.M.): amber, acid; specific gravity 1022; slightly plus normal trace albumin; centrifuged twenty-five minutes; red blood corpuscles scattered in all fields, from two to three to eight to fifteen; many fields contained over a dozen; a few groups of from four to twelve, with a fair number singly; leukocytes in moderate number; stray cylindroids and hyaline casts; few granular cells, twice the size of polymorphonuclear leukocytes. *x*-rayed twice by Dr. Leonard. Negative.

Now for a few days no severe attacks occurred. At the present writing (April 20th) pain has returned as before, and marked sensitiveness exists anteriorly from the loin to the pubes, but especially along the middle course of the ureter. The pain as described has occurred from just below the false ribs to the pubes. Loin sensitiveness to pressure in this examination, but not so markedly as in the middle situation of the ureter.

This case is regarded as either one of renal stone or as ureteritis. There are no indications of tuberculosis or of renal neoplasm. It resembles in character that of the two preceding.

There is a rather extensive anteroposterior diameter of the abdomen. Leonard was unable to outline the kidney in the skiagraph, but from his negative finding regards stone absent. An exploration of the kidney is under consideration.

A case resembling the foregoing is the following: In this case the diagnosis lay between stone in left kidney or presence of a neoplasm. The patient was lost sight of after twelve days' observation.

CASE IV.—L. J. F., aged sixty-three years; clerk; first seen June 16, 1902. Referred to me by a colleague for presumed gastrointestinal disorder. For four or five years he had had a good deal of distress in the abdomen, in the region of the descending colon, which for the past few months had been greatly aggravated, and for a few weeks it had practically incapacitated him. Continued soreness, with attacks of severe colicky pain, lasting from a few hours to two or three days; pain usually starts with a griping sensation in the region of the sigmoid and ascends to the loin, but is not felt laterally or posteriorly. It may be so severe that unless relieved he has a sensation of impending death during the seizures. The passage of flatus promptly relieves him, as does, too, but to a less extent, the application of heat; no frequency of urination; bowels much constipated. Eight years before passed a tape-worm, after extended and futile treatment. Then had pain in region of the sigmoid for a long time.

Physical Examination. Thick-walled abdomen; sensitiveness along region of descending colon, and laterally in the loin, but not

in the loin posteriorly; impaction of feces; arteriosclerosis; heart normal; accentuated second aortic sound at apex.

Examination of stomach contents shows high-grade hyperchlorhydria; large high enema, and castor oil by the mouth ordered. An enormous quantity of hardened feces passed. Obstruction of the colon by neoplasm or simple stricture was thought of in the first examination. However, 1500 c.c. of fluid were readily passed into the bowel without pain or distress after it was first emptied, and subsequently the bowel was inflated without discomfort. Colon and sigmoid tympanitic; no abnormality detectable; despite thorough evacuation of the bowels attacks of pain occurred as before, and sensitiveness in the loin laterally, but not posteriorly, noted. Pain shoots into the left testicle.

Urine Examination June 16th. Acid; amber; specific gravity 1018; well-marked trace of albumin. Microscopically: all fields crowded with red blood corpuscles; numerous leukocytes. Centrifuged specimen showed blood sediment. Specimen June 28th, voided in laboratory: clear, light amber; acid; no visible sediment; albumin, moderate trace. Centrifuged specimen: all fields crowded with red blood corpuscles; few narrow hyaline casts; granular debris; stray leukocytes. On careful inquiry fact elicited that five weeks prior to his first visit urine on one occasion was noted to be very dark, presumably bloody.

The patient was unwilling to undergo the expense of an *x*-ray examination by a competent man, and would have refused operation if stone had been thus detected. He was under observation only twelve days. Since then I have been unable to locate him.

Of these 4 cases, operation disclosed stone in 2, as was previously diagnosed. In the 2 last described there is little doubt that the symptoms also had seat in the left kidney or ureter, but whether the disorder be stone, a ureteritis, or a renal neoplasm it is now impossible to determine. It is of especial interest that in all these cases the symptoms were more or less referred from the offending organ to the bowel. In but 1 of the 3 did I suspect stone in the initial visit. In the first of these cases described I was originally completely deceived as to the nature of the ailment, and only examination of the urine suggested it. In Case III. several visits were made before the likelihood of the kidney being the seat of the trouble occurred to me. In Case IV., also, it was only after attention to the bowels, excluding disease here, that the notion of stone or another gross kidney lesion was considered. It is especially interesting that without examination of the urine in the first of these cases of stone the correct diagnosis would not only have been impossible, but would probably not have even suggested itself.

I wish especially to call attention to the chief location of pain in 3 of the cases, which was also manifest, though to a less degree, in the fourth (Case II., in which stone was diagnosed and removed),

an area of extreme sensitiveness to touch and of unprovoked pain below and to the left of the umbilicus, at about the site at which the ureter crosses the pelvic loin. I have nowhere seen this symptom noted, and my experience in these, and in other cases of stone with frank symptoms, suggest it may be of value in diagnosis.

In Case II., in which the influence of posture on the pain was very marked, and in which, as in Case III., the pains were pronouncedly aggravated at night in recumbency, the pain was referred anteriorly along a portion of the ureter covered by the sigmoid flexure (see diagram, Case II.). The diagnosis in this case was the least difficult. At the initial visit, after eliciting the history, and before making a physical examination, I suspected stone, and so informed the patient. Here the unvariable aggravation of symptoms through a dietetic error, which coincidently induced bowel flatulence, and the marked relief in pain after the passage of gas, totally misled his former attendants.

Of these cases, as remarked, operation disclosed 2 to be renal calculus. The second 2, unoperated on, may be here grouped with them, as, if not those of stone, at least due to a unilateral renal abnormality simulating it. The surface symptoms of all were outside and away from the kidney, thus placing them in the category of so-called "masked" calculus. Whether 2 of these be stone is less material than that a study of the cases at least made clear the location of the ailment in the kidney previously regarded as having seat in the intestines.

A condition termed arteriosclerotic abdominal pain has lately attracted considerable attention, and there is now on record a number of these cases[1] in which, as described by Buch, as a result of a sclerosis of the vessels in the splanchnic area, the chief symptom is recurring abdominal pain. The pain is paroxysmal, of frequent recurrence, and is induced by the factors exciting anginal seizures with which it is in no one group associated. The pains have their seat almost always above the umbilicus, and are only exceptionally associated with digestive disturbance or meteorism. It is with such cases that the second 2 of the above reported might be confused, since in these, with attending renal fibrosis, the frequent presence of red blood cells in the urine could not be regarded as unlikely. The first of these cases (H. M.) in the initial examination, with no cause for pain evident, I was inclined to place in this group. The ages of each, fifty-five and sixty-three years, and the presence of indications of general arteriosclerosis were in its favor. But the invariable one-sidedness of the pain, its situation below the umbilicus, and the presence of distinct digestive (bowel) disturbance with combined marked meteorism, plus the indications of unilateral renal disorder, opposed this view.

[1] Max Buch. St. Petersburger med. Wochen, 1904, vol. xxvii., has recently collected and analyzed 25 cases from literature and from his own observation.

It should be remarked that in the diagnosis of renal calculus no stress is laid on the mere presence of microscopic blood in the urine, without other discoverable symptoms of unilateral kidney disease, since it is not unusual for red blood corpuscles to be more or less constantly found in cases of chronic nephritis, even with the scant presence of casts, and their occurrence through tubule irritation in oxaluria and lithuria is well recognized. It is their more or less persistent presence with symptoms which may be intelligently interpreted to indicate unilateral kidney affections, whether stone or a lesion of another sort, that is significant in this particular, as it was in the four cases just detailed.

Frank renal hemorrhage, in cases in which both the symptoms and a cystoscopic and ureteral examination show to be unilateral, may occur without the presence of calculus, as it may without apparent evidence of a gross anatomical alteration in the kidney. In Eshner's[1] review of the literature of unilateral renal hemorrhage he cites several interesting instances, and more recently H. Schüller,[2] in a "Contribution to the Study of Hemorrhage from Apparently Normal Kidneys," reports a case of recurring and obstinate "unaccountable renal hemorrhage," shown to proceed from the right kidney, in which, by exploration and the usual autopsy incision, no gross lesion was found, but in which an examination of a bit excised showed evidence of an old inflammatory process. In a review of the literature of unconcealed unilateral renal hemorrhage, in cases in which gross lesions were regarded as absent, he finds two which were not evidently pathological, and these he prefers to class as "unexplained," rather than to permit such to be described as hæmaturia without lesion, "local hæmophilia" (Senator), "essential hæmaturia,"[3] or "angioneurotic hæmaturia" (Klemperer), "hemorrhage from healthy kidneys,"[4] The various pathological findings in these cases he groups as congenital malformations, diffuse unilateral nephritis, glomerulonephritis, fibroid degeneration of the fatty capsule, mechanical lesions and their effects, and a growth including such a case as Abbé[5] described, in which the hemorrhage apparently arose from a calcareous incrustation of a papilla.

He holds that in cases not so included careful examination should practically always reveal some organic lesion, however insignificant.

But the likelihood of the existence of undiscovered stone in certain of these cases is not sufficiently excluded. It has only in the past few years begun to be generally understood that by mere exposure and palpation (in Eshner's case of unexplained unilateral hæmaturia the kidney was exposed and manipulated, but not explored), and even repeated needling[6] without section, stone cannot

[1] Loc. cit.
[2] Wien. klin. Woch., 1904, Bd. xvii. p. 477.
[3] Nitze. Vereins-beilage der Deutschen med. Woch., February 18, 1897, p. 54.
[4] Deutsche med. Woch., February 25, 1897.
[5] Medical Record, May 16, 1891.
[6] As is remarked by Morris, Renal Surgery, p. 118, "needling the kidney for diagnostic purposes is most unreliable and should be abandoned."

be excluded, and the likelihood of a calculus of small size being overlooked by the usual incision of the kidney and exploration of its pelvis been apparent. This difficulty is well illustrated in a case reported by Abbé,[1] in which, but for the previous unmistakable findings, a presumed thorough search for a calculus in an incision made through the greater curvature of the kidney into the pelvis would have been finally abandoned as resultless, and the case classed as one of simple pyelonephrosis. Similar cases in the past few years have come to my notice. A study of paraffin casts of the pelvis of the kidney and the relation of the pelvis to the calices, made in the laboratory of the Episcopal Hospital some years ago by Dr. Ghriskey, graphically showed the irregular branching of the latter, and explained the difficulty often encountered in finding even an unencysted small stone. It demonstrated to us the necessity for a section of the kidney of sufficient size that all parts of the pelvis may be explored, and by a route to the kidney which also permits of ready access to the ureter.

I wish now to describe three cases of unusual interest, the symptoms of which suggested stone in the kidney, and in two of which the correct diagnosis was impossible without exploration of the kidney. In the first case to be related, which occurred before the advent of the Roentgen-ray method, there were certain of the usual symptoms of calculus, hæmaturia, both frank and larvated, with recurring attacks of renal colic. Death occurred from acute nephritis, complicating an attack of influenza, while the case was under observation. A necropsy revealed a strictured ureter, which, causing intermittent hydronephrosis of small degree, with hæmaturia, originated the symptoms.

Ureteritis as a cause of simulation of renal calculus is of special interest. It is, as remarked by Morris, a disease which has attracted more attention in Germany and France than in England or the United States, and has been especially studied by Israel. Case II. of this series is probably also one of this sort, a ureteritis existing as a part of the general perinephritis found.

The occurrence of hæmaturia is especially interesting in the case to be described as a symptom of slight intermittent hydronephrosis due to ureteral obstruction. There are a few of these cases on record. Bangs,[2] in a search of the literature, was able to collect but 13 cases in which hydronephrosis was associated with hæmaturia.

CASE V.—G. W. B., aged twenty-six years. First seen August 3, 1894. Patient was referred to me for examination by Dr. W. W. Keen, with a diagnosis of probable stone in the kidney. He had been in good health until five years before. He then, without noticeable premonitory symptoms, had an attack of severe pain in the

[1] Annals of Surgery, August, 1899, p. 186 *et seq.* Observations on the Detection of Small Renal Calculi by the Roentgen Ray.

[2] Medical News, February 11, 1905.

right loin. It was of precipitated onset, and of about ten hours' duration. After a two months' interval of freedom, a second similar attack occurred, associated with profuse hæmaturia, and this seizure was followed by others without noticeable hæmaturia, at intervals of from four to six weeks for the first year, about three weeks' intervals during the second, and during the following three years had attacks with an interlapse of one to two weeks. In the year preceding they had been less frequent, not over six occurring at intervals of from one to three months. He had noticed it usual that, when occurring at about weekly intervals, the seizures were not so severe as when a longer period intervened. The onset of nearly all the attacks, save the first, was marked by the early occurrence of a dull aching in the loin, not existing previously, and accompanied by chilliness without a rise in temperature. The pain, increasing in severity, reached an acme in a few hours, and so continued for some hours, and then gradually declined, the dull ache or soreness commonly remaining for two to three days. The fixed pain which occurred in the loin with the seizures was not felt laterally or anteriorly, and occasionally radiation along the ureter to the bladder was noticed; and in a seizure he had in a day or so after he first saw me there had been pain felt in the head of the penis. A moderate degree of bladder irritability during the attack was common. Vomiting was usual during the height of the seizure. The appearance of the urine, save on the occasion of the second attack, had not suggested to him that it contained blood; gravel in some amount he had occasionally noticed. He had never remarked increase in the amount of urine during the subsidence of an attack, nor diminution preceding, nor a sensation of fulness in the loin to suggest a hydronephrosis. No pain was present in the intervals, and no tenderness existed on manipulation of the loin in these examinations.

During the early period of the seizures the condition of the stomach seemed to influence their occurrence. Errors in diet would, he said, precipitate an attack. This relation he had not, however, noticed very lately.

The patient had seen several eminent medical consultants with Dr. Marcy, his attendant, but, despite the treatment, his ailment continued. He was referred to me by Dr. Keen, in order that a careful study of his case be made and a course of medical treatment instituted prior to resorting to operation, to which the patient was then absolutely opposed. The patient was noted to be of spare build; height, five feet six inches. His present weight was 113 pounds, a loss of about twelve pounds from his best five years before. His general health was fair, apart from the occurrence of these seizures. He had never sustained an injury or strain to the back or loin in any manner that he could recall. Physical examination of the abdomen revealed nothing abnormal, except that at bimanual palpation there was a somewhat increased resistance on the right

side. There was no indication of tumor, nor was the kidney distinctly palpable. Tenderness was absent.

He had previously lived in a malarial district, but never had had chills or fever. Repeated blood examination showed no plasmodia. Several examinations of the urine in the intervals, and one during an attack, all showed red blood corpuscles in large numbers, clumped and single; pus cells in moderate number; free uric acid in abundance (in freshly voided, centrifuged urine), and on one occasion only calcium oxalate. (This examination was made eighteen hours after voiding.) Casts were always present: hyaline, coarsely and finely granulated, broad and narrow, and, sparingly, epithelial casts; cylindroids always abounded. Albumin was present in moderate amount. Tubercle bacilli were carefully searched for over some time resultlessly. Specimens voided during and at the termination of a seizure were especially rich in red blood corpuscles and in pus cells. Twenty-four-hour specimens were collected over some time, to assist in determining whether there were sufficient variation to suggest obstruction of the ureter. But there was practically no variation over a considerable period, and a constant watch for indications of flush-tank symptoms was resultless.

Considering all the facts in the case, Dr. Keen and myself regarded the diagnosis of stone in the kidney as probable. The patient was given piperazin in doses of ½ drachm daily, which he took for about three months, alternating it at short intervals with lycetol in similar doses. Curiously, after a few weeks' trial of the remedy, the attacks diminshed in severity, and were apparently not so readily induced by exertion as formerly, and he regarded himself as decidedly improved. With this view, however, I did not coincide, and repeatedly urged on him the necessity of an exploratory operation, as the presence of blood showed no material change, but his apparent improvement made him even more averse than at first to operation. During October and November he travelled a good deal, and was infrequently seen. On October 8, 1894, Dr. Marcy reported that he was very ill with influenza, complicated by acute nephritis; that his temperature had run very high, and that his urine, which at first was voided in full amount, contained much albumin and many casts. On the second day of the disease the urine was visibly bloody and became diminished in amount. The patient lived in another town, and I saw him but once after this with Dr. Marcy. Under active treatment improvement occurred, and secretion of the urine was re-established. On the seventh day of the disease, while apparently doing well, a severe rigor, followed by total suppression of the urine, occurred, and he went into a condition of collapse, during which death occurred. His mind remained clear throughout the attack up to the time of his decease.

The post-mortem examination made by Dr. Marcy was limited at the request of the family to the kidneys. The right kidney was

enlarged and adherent to the adjacent parts. The ureter was strictured at a point two and a quarter inches from the pelvis. This constriction extended for an inch. An ordinary probe, No. 3 of the French catheter scale, would pass, but a probe of No. 8 size would not pass the obstruction (Dr. Keen). The renal pelvis and the ureter above the constriction were dilated; the pelvis contained from four to six ounces of bloody urine. The left kidney was also reported by Dr. Marcy to be much enlarged and to be "the seat of acute inflammatory change." The specimen was given to Dr. Coplin for pathological examination, but, unfortunately, it was mislaid without other than the above observations of Dr. Keen and Dr. Marcy being recorded.

In the following case the diagnosis was in doubt for some time. The subject was highly neurotic. The symptoms were largely gastrointestinal. There were recurring attacks of obstinate and severe gastralgia and enteralgia. A careful study of the case caused me to place the diagnosis between stone in the kidney and ureteritis, but in favor of the former. Operation by Dr. Keen disclosed a kidney apparently healthy, so densely bound by adhesions that its delivery through the wound was impossible; it was likewise impossible to gain access to the ureter. The adhesions interfering, as they probably did, with the function of the kidney and the ureter, were doubtless the cause of the symptoms. The frequent presence of microscopic blood in the urine and the finding of streptococci suggested stone, probably associated with a (microscopic)[1] pyelitis.

CASE VI.—C. H., aged thirty years; milk dealer. The history elicited was that he had been in good health until seven years before. Then a condition of mental and physical unrest developed, without assignable cause, and he "had to keep constantly on the go." About three years later he had a seizure of a curious character. He was nervously apprehensive on rising in the morning, and this continued through the day; there appeared a sensation of numbness over the whole of the left half of the body, which remained for several days; with this, and also continuing for several days, there was a good deal of abdominal cramp. He was thus confined to the house a week. A second seizure of this sort did not occur until June, 1898, (three and a half years later). The third appeared again in two months; and since then the seizures occurred at intervals of two to four weeks, and lately about two weeks apart. Most of these later seizures were as follows: Crampy pain began a little above the umbilicus, and thence radiated over the abdomen; the pain was of greater intensity above than below the umbilicus, and more severe to the left than to the right. Colicky pain lasted steadily three to four days, and he was commonly housed a week by an attack. During a seizure the abdomen was rigid, but not especially distended by gas, nor

[1] Pus cells were always in less amount than were red blood corpuscles.

tender on pressure. Pressure rather relieves the pain, as is usual in paroxysms of lead colic. The attacks necessitated a free use of morphine hypodermically. He had lost fifteen pounds in the past year or two.

Physical Examination at the Date of First Visit. Chest: Heart normal, first sound lacking in tone; lungs normal. Abdomen: Liver and spleen, outline normal; neither kidney plainly palpable; stomach slightly dropped. Fine tremor about the lips and in the tongue on protrusion; purplish line on gums; knee-jerks exaggerated; station and pupils normal; skin of sallow hue. Is an excessive tobacco chewer; habits otherwise good. No lues. No neurotic family history obtainable. No source of lead poisoning evident.

Later, the following was elicited: Attacks nearly always occurred between 10 P.M. and 2 A.M., but one recent seizure had occurred at 6 P.M., lasting two days. This came on before he had eaten supper, and while his wife was in the throes of confinement. It occurred while he was sitting talking with the nurse, and in no way previously nervous or excited. The pain came on gradually, reaching its acme in one and a half hours.

No symptoms of indigestion were prominent, and he has not noted (as in the case of K.) that a dietetic error precedes the attacks. The bowels were commonly constipated. Examination of the stomach contents showed practically normal secretory power and some impairment of motility, with imperfect, starchy transformation. Blood examination for plasmodium, negative. Urine examination first made, negative.

The diagnosis by a process of exclusion was at first ordinary gastralgia in a neurotic subject. Treatment directed to improving the gastric myasthenia and the general physical and nervous tone was efficient. He gained in weight and improved in appearance, and the attacks were of less frequent occurrence. After a few weeks' treatment he passed for a time from observation. In July, four months after he was first seen, it was noted that in the attacks, which still occurred, pain began in the centre of the abdomen, at first slight, but soon augmented so in severity as to necessitate the use of morphine. Pain diffused from the centre of the abdomen to the left hypochondrium, and then into the back over the region of the left kidney, limiting itself at first to these two situations, and sometimes radiating toward the pubes, but finally diffusing over the whole abdomen, but lower and more intensely on the left than on the right side. He thought the urine was frequently scanty in amount during the seizure, but frequent desire to void the urine was not common. At this visit I began to suspect the likelihood of renal calculus, and then arranged for an *x*-ray examination. The specimen of urine then examined was negative; no albumin and no red blood corpuscles or leukocytes noted. On July 1st it was remarked that for the past few days he had had considerable pain in the left loin through the

back, and that he had not been able to lie without discomfort on the left side. Urine examination again negative. *X*-ray examination by Dr. Leonard negative as to stone.

October 3d: my case-book notes four severe attacks since the preceding June, each of from three to five days' duration. In two of these the deep-seated, dull ache in the left loin developed as the pain anteriorly ceased; this continued for several days (three to four) and was of pronounced severity. To ease it he was compelled to lie during this time with a soft pillow under the loin. On another occasion pain occurred along the course of the lower part of the left

FIG. 6

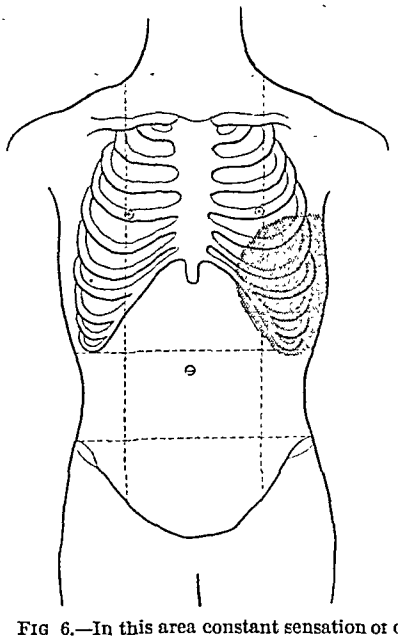


FIG. 7

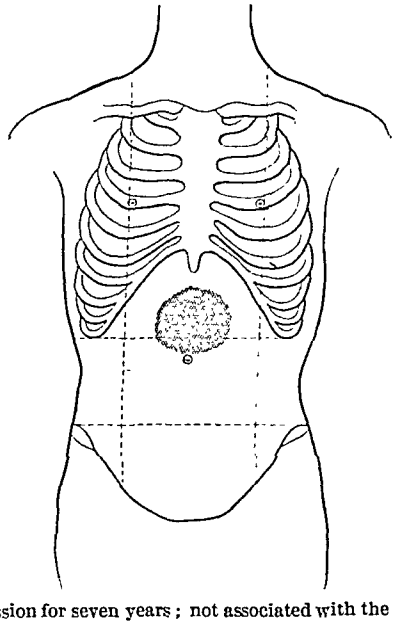


FIG 6.—In this area constant sensation or oppression for seven years; not associated with the attacks of cramp.

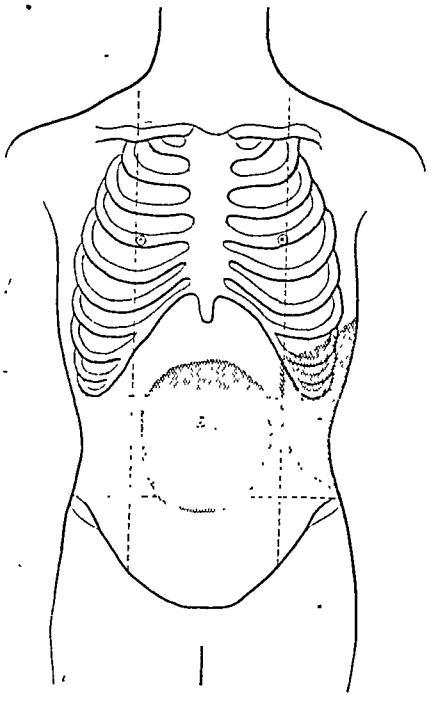
FIG. 7.—This exactly represents situation of pain in attack on October 11th and 12th. None in any other situation.

ureter. In all the attacks pain was felt in the left hypochondrium, extending into the back.

On October 11th an attack occurred, the pain of which was centred in the epigastrium above the umbilicus, and did not extend into the back or loin. On November 4th an attack of four days' duration, about forty-eight hours (severely) in the epigastrium, and (lightly) for nearly the same period in the left loin. On January 9th it was noted no attack since the above, but constant slight or considerable distress in the left hypochondrium, with a sensation as if a heavy weight occupied this region. Urine then voided in laboratory showed scattered red blood corpuscles, and in two flakes a great

aggregation of them, together with polymorphonuclear leukocytes. Some twelve examinations of the urine were made from October to the day of the operation. Three of these were voided during an attack. In the latter red blood corpuscles in fair amount were found, more numerous in specimen during and at the termination of an attack. Pus corpuscles, single and in groups, were noted. Mucous and hyaline cylindroids, hyaline casts, and a fair trace of albumin were usually evident, as were on several occasions hyaline casts carrying red blood corpuscles and granular epithelial cells, evidently from the renal tubules. Some four of the specimens were voided in my presence, the urine flushing the urethra being discarded. Crys-

FIG. 8



Attack lasting forty-eight hours. Onset in centre of abdomen; pain so severe as to "double" him up, diffused to left loin, to the lower dorsal and lumbar region, and left hypochondrium, and finally over whole abdomen.

talline elements were rarely seen in the freshly voided specimens. Only once, and that after rather severe muscular exertion, purposely undertaken for the examination, a glass of hot water being previously drunk and the bladder previously emptied, were free uric acid crystals numerous. Then, as on other occasions, there were a moderate number of red blood corpuscles and a few hyaline casts. Calcium oxalate crystals were never seen. On several occasions there were noted present in the suspended mucinous sediment, which developed when urine had stood a few hours, a few whitish flakes; these were made up of aggregation of pus corpuscles, with red blood corpuscles and moderate number of ovoid nucleated cells, twice or

thrice the size of polymorphonuclear cells. There were also twisted mucinous casts, as if from the ureter, which, on addition of acetic acid, were found to be made up chiefly of polymorphonuclear leukocytes. Chains of streptococci were also noted in a specimen examined by Dr. Ghriskey.

Operation by Dr. W. W. Keen January 18, 1900. Kidney unusually difficult to reach and impossible to deliver because of dense adhesions binding it to adjacent parts. Section and thorough exploration through the wound; no stone detected. Repeated but ineffectual efforts were made to gain access to the ureter. The kidney did not appear especially enlarged, nor its pelvis dilated. Dr. Keen stated that no blood was noted to be passed in urine after operation, which suggested obstruction of the ureter.

Recovery from operation without complication. Dr. Minich reported to Dr. Keen that on April 1st, ten weeks after operation, patient had the "worst attack he had ever had," requiring ¾ grain of morphine, hypodermically, for its relief, and again in June two severe attacks, "the seat of pain being a little to the left of the umbilicus." He has not reported to Dr. Keen or myself since the operation, and repeated efforts since the above report to locate him have been unavailing.

A second exploration of the kidney and by the route and method adopted by Morris, the oblique lumbar,[1] was here regarded as essential, in order to gain free access to the ureter, which the indications are is the seat of partial obstruction from a ureteritis accompanying the perirenal inflammatory process.

In the case to be described the chief points of interest are simulation of stone in the kidney in a neurotic; the subject of severe attacks of left-side loin colic; resultless exploration of the kidney; a second section proposed; a further study excluded stone. Prompt recovery followed a change in the patient's mental condition.

CASE VII.—J. P., aged twenty-five years, married, white, American, clerical work, consulted me February 12, 1901; the chief points in the history then elicited were:

The first symptoms of his ailment appeared in June, 1899, with an attack of left-sided renal colic of eight to ten hours' duration; he stated a small stone voided; has had recurring attacks of left-sided loin pain since then from once to twice a month, and nineteen such stones observed by him to have been voided. He has recovered several of these, but had, he stated, given them to one of his previous attendants.

In September, 1899, he had gone to the Johns Hopkins Hospital, where he had been an in-patient for some weeks, and was later transferred to the surgical side. He said that he had been *x*-rayed, a large stone noted, and that Dr. Halsted had operated in January,

[1] Renal Surgery, Hunterian Lectures, 1898, p. 116.

1900, removing a calculus the size and shape of the thumb, but that ten days after operation the pains recurred as before. Since operation, three or four stones passed, following eight to ten severe attacks. Attacks were all very similar. Suddenly appearing severe left-sided loin pain, approximately from loin to abdomen, but no tendency to shoot toward bladder, testicle, or thigh. Commonly nausea and vomiting at the height of attack; sometimes delirious; usually requires morphine hypodermically for relief; sitting posture, "chin to knees, and head to the left, helpful." Five years before he had had severe attacks of colic, forty hours' duration, in the same situation. Had several hemorrhages, it was thought from the lungs, about this time, but a New York consultant had said from the stomach. No other past or present indication of lung trouble. Has had "chills and fever" every summer for a number of years. Lived for many years in the South. Has had the diseases of childhood; in adult life pneumonia twice; dysentery for six weeks; several accidents; operation for double hernia, January, 1898; no syphilis; no family history of tuberculosis or carcinoma; moderate alcohol, but excessive tobacco (cigarettes) indulgence; coffee in excess.

Physical Examination. Lungs normal; heart irritable (coffee and tobacco); no valvular lesion; neither kidney palpable; spleen not enlarged; liver outline normal; stomach slightly myasthenic; inferior border to umbilicus; scar from past hernia operation visible.

Blood examination for plasmodia, negative.

Urine Examination. Voided in laboratory day of first visit, clear, colorless; specific gravity, 1002; no sediment for microscopic examination; albumin absent; February 13th, day specimen: 180 c.c., clear; light amber; acid; specific gravity, 1015; small trace of albumin. Centrifuged specimen: scanty sediment; scattered few uric acid crystals; stray red blood corpuscles and leukocytes. February 26th, day specimen: colorless; specific gravity, 1003; no sediment,

In response to inquiry, Dr. Halsted wrote me that he had operated for stone, which he had failed to find, though he thought he had explored the pelvis and calices of the kidney very thoroughly, but that he now could understand from later anatomical study of the kidney how one or more stones might have been overlooked; that no *x*-ray examination had been made; it had been arranged for with Dr. Leonard, Dr. Halsted going to Philadelphia for that purpose. The patient had failed to keep his appointment, instead had gone off and got married.

Dr. Halsted regarded it likely that a stone (or stones) was present and favored a second operation. The Johns Hopkins Hospital medical record of the case prior to operation was interesting. The following is briefly noted from it: He had been in the hospital from September 3 to 29, 1899.

Diagnosis: renal colic. Lately sleeplessness; anorexia; palpitation;

flatulency; headache only since doing office work (eye-strain); tendency to moist hands and feet; very active dermatographia; a recent history of dysentery; frequent attacks of "chills, high fever, and sweats on alternate days;" said to have been cured by quinine.

Physical examination disclosed no abnormality in chest or abdomen; kidneys not palpable; a series of attacks was observed in the hospital. Chief points noted in first attack were discomfort in the umbilical region, thence to left loin, increasing to pain; very severe; vomiting relieved; no radiation to testicle; no retraction (in one attack retraction noted); no pain on pressure in region of kidney; slight sensitiveness; kidney not palpable. After some hours, the paroxysm continuing, pain suddenly ceased and 1000 c.c. urine voided "in which a little blood evenly distributed, not apparent until after standing." No attempt to micturate during the attack; free from pain for forty-eight hours. 6 P.M., a second seizure. Discomfort in abdomen and loin; two hours later "vomiting and great pain in left lumbar region; hot bath and morphine; no relief; during spasms of pain delirious." "Pains seemed most severe, but patient afterward had no consciousness of it." "Attack ceased about 3 P.M.; uneasiness continued until 9 A.M. Vomiting frequently preceded spasm or occurred during seizure. Toward the end of series of attacks marked neurotic element." "Voided no urine since 6 P.M. last night (September 12th) until 3 P.M. Then 1200 c.c.; no blood; no gravel." "September 17th, patient has had a number of attacks very similar to those of September 12th. Early during the attack 1000 c.c. urine voided. After passing a considerable quantity flow suddenly stopped. In about fifteen minutes a little muscular spasm felt, but no pain; then a few drops of blood from penis. Flow of urine again natural. Testicles retracted; attempts to micturate once or twice during the attack unsuccessful."

Three records of urine examinations: September 9th: "1000 c.c., clear; neutral reaction; pale yellow; specific gravity, 1007; trace of albumin; cloudy sediment; stringy pink; red blood corpuscles numerous." "September 10th: 1040 c.c., clear; amber; faintly acid; specific gravity, 1018; mucus cloudy; calcium oxalate in clumps; occasional pus cells. September 17th: 1040 c.c., turbid; pale yellow; faintly acid; specific gravity, 1012; albumin, trace. Sediment, mucous cloud; red blood corpuscles and pus cells numerous. Exploratory nephrotomy: Section left kidney; thorough exploration of pelvis. No stone found; no abnormality noted. Typical attacks of renal colic."

I regarded the case as probably renal calculus, which had been overlooked in operation, and in this opinion Dr. Halsted himself, in a personal letter, coincided. Two skiagraphs taken by Dr. Leonard were negative. Patient admitted to my service at the Episcopal Hospital for observation. Here he remained several weeks in a private room. Out-of-door privileges. A number of observed

attacks were similar in outline to those noted occurring at the Johns Hopkins Hospital; seizures lasted from a few hours to a day or two; pain apparently of intense severity in some; at times then delirious. Urine suppressed for twelve hours; then a large quantity of very low gravity voided. Morphine demanded for relief of pain, but hypodermic injections of water alone in later ones efficient. States he passed several small stones in the public urinal during sojourn at the Episcopal Hospital, which he could not recover. Many urine examinations were practically similar to the foregoing. At the end of seizure, volume usually large. Gravity low. In interval, average gravity 1015 to 1021; usually clear; amber; acid; and occasionally stray red blood corpuscles. Tubercle bacilli looked for resultlessly. On one occasion, shortly before he left the hospital, and on another day after he stated he had passed a small stone, specimen of urine asked for by Dr. Ghriskey in hospital laboratory. The patient took the glass into an adjacent room; specimen furnished was visibly blood-stained. Dr. Ghriskey noted a drop of bright-red blood on the patient's handkerchief. It was believed he had pricked his finger and introduced blood into the urine. Several blood examinations (from finger) had been made in this case, and he had been in the habit of watching the work done in the laboratory and often seen blood so taken from patients for examination.

There is a record of twelve urine examinations made by Dr. Ghriskey, of single specimens, and of the twenty-four hours' urine during the seizures, at their termination, and in the interval. In all the centrifuge was employed; but twice were (stray) red blood corpuscles noted. On two occasions the urine was markedly bloody, but in the light of present knowledge it is believed, as above remarked, to have been tampered with.

Despite the negative finding of the *x*-ray, Dr. Halsted, who was communicated with on several occasions while the patient was in the hospital, believed stone present, and favored a second exploration. This the patient was apparently agreeable to; it was in contemplation when he suddenly, without previous notice, disappeared from the hospital, leaving his wardrobe, including even his watch, and he was not heard from for some time.

Certain features about the seizures, a strong neurotic element frequently manifest, including the relief obtained during the attacks by water hypodermics; the curious retention of urine for a day or so without discomfort; the passage of a large quantity of a gravity approaching water; many discrepancies and inconsistencies and actual attempts at deception in his statements as to past and present attacks, including the asserted passage of calculi, had caused us in the hospital to not only recognize a strong emotional element in the case, but to question if that were not the whole feature. The subsequent history practically demonstrated the correctness of this view. The patient was dissatisfied with his previous clerical occupation,

and there were several elements in his domestic life that would readily serve to aggravate a neurotic tendency. He had an opportunity through influential friends to enter the regular service of the army, in which he had during the Spanish War been a volunteer officer, and the life appealed strongly to him. On leaving the hospital he had gone to Washington; successfully passed the civil service and the medical examination, and received an appointment. I lately heard from a kinsman of his who had originally sent the case to me, that he had been constantly in active duty, and from the time of his appointment had remained in good health, and was totally free from these attacks.

This case has been narrated somewhat in detail because of its unusual features. Here apparently pure hysteria so mimicked the features of renal calculus that his many attendants were deceived. An exploration of the kidney had been done with his ready acquiescence, and the second had been contemplated. The attacks of unilateral colic were undoubted, but they were apparently, as the later history showed, an expression of hysteria. To give verity to and emphasize the ailment which the patient himself regarded as kidney stone, he had wilfully misled in stating that he had passed calculi, as he had attempted to deceive in first relating his history—that he had been *x*-rayed while under Dr. Halsted, and a large stone seen, describing the shape and size of the suppositious stone removed. He is believed to have similarly deceived on more than one occasion in introducing blood into the urine specimen for examination.

CERTAIN HYPERTENSIVE CRISES IN ARTERIOSCLEROTIC SUBJECTS.

By John Bradford Briggs, M.D.,
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After eliminating all cases showing the more profound changes of general arteriosclerosis, all those of gross cardiac lesions, and those of manifest interstitial nephritis, there remains an instructive group of individuals, presenting many and varied symptoms, but only one definite or constant physical sign—that of hypertension of the pulse. The patients to whom I refer, coming from every class in society, of various occupations and of every diversity of personal habit and habits, are alike in virtual age. They are all nearing or have passed a certain grand climacteric; their footsteps have crossed the Simplon of the vital Alp, and are definitely tending toward the grave. Their actual count of years may be many as we reckon them, or few as compared with the improved chances of life expectation tables, but one and all they come to us to tell of the first fore-

shadowing of their far-off but approaching fate. The signal, whatever it may be and whatever its form, is the "tap on the shoulder" of physiological old age, nature's warning of the lesser death. To some it comes as the bill for a long life, to others as that for a hard one, while to another group it is but the expression of defective vascular heredity—from one and all it demands an account for much life lived, and signifies that not so much is left.

With certain of the forms of this universal signal we are very familiar. The pipe-stem arteries, the whitening hair, the joints losing their suppleness and the muscles and nerves their agility of function, speak with equal distinctness to the layman and to his physician. Certain others are appreciated only by the medical man—especially that permanent trace of albumin and those few tube casts that may be of such an advantage to a man when they are properly interpreted at the right time.[1] But very, very often, when nature is in a kindly mood, she speaks in still milder accents, and her softly whispered "Go slow!" is uttered at an even earlier period. Early enough, if the admonition be heard and heeded, for the period of physiological old age not only to be prolonged, but even to be avoided for many years. It is of this admonition, when it takes the form of expression of hypertension of the pulse, and with some of its relations to albuminuria, that I wish to speak.

That there is an intimate relation between pathological states of high blood pressure and renal permeability to albumin is possibly a notorious clinical fact. Numerous observations on widely different classes of cases—infectious, cardiovascular, renal—early convinced Cook and the author[2] that if hypertension is not the inevitable forerunner and companion of albuminuria, at least in any individual the clinical gravity, if not the actual measurable degree of his albuminuria, is generally roughly parallel to the line of elevation of his blood pressure. To claim that every albuminuric event in the previously healthy, or every increase in albuminuria in those whose kidneys are already intolerant, is necessarily preceded by a dramatic rise in blood pressure, and immediately caused by it, would not meet the facts.[3] But that in the majority of patients whose albuminuria is at all a cause for anxiety, the clinical measurement of blood pressure is capable of furnishing an early danger signal, and even of serving as a handmaiden to the successful therapeutist, is a statement easily susceptible of proof. Janeway[4] recognizes the emphatic importance of hypertension in connection with the contracted kidney, and gives a striking illustration of its worth as an aid in a differential diagnosis. It is not so much with cases

[1] Osler, New York Medical Journal, November 23, 1901.

[2] Cook and Briggs, Clinical Observations on Blood Pressure, Johns Hopkins Hospital Reports, vol. xi.

[3] Erlanger and Hooker, American Journal of Physiology, 1904, vol. x., Proceedings of the American Physiological Society, p. 16.

[4] T. C. Janeway, The Clinical Study of Blood Pressure, 1904, p. 172.

of this type that these notes will have to do, as with another clinical group, the vascular origin of whose renal lesion is beyond question, and in whom vascular and vasomotor changes may be followed, as they determine variations in the state of the kidneys. However clear as pathological entities the "true granular" and the patchy red arteriosclerotic kidney may be, and however certainly their urines may be recognized and even differentiated from one another, it is far from true that every arteriosclerotic individual, whose urine at a certain examination shows a trace of albumin, is suffering from one or the other of these gross lesions. It is understood, of course, that we are not referring to the acute febrile, nor the cyclic or other albuminurias of a transient nature. Entirely aside from such conditions I believe we may detect traces of albumin in the urine at a time when the kidney has suffered no irreparable structural damage, and when its impermeability to proteids in solution may perhaps be fully restored. The prenephritic stage, if the term may be permitted, is never so frequently recognizable as in the subjects of generalized arteriosclerosis. In the absence of any profound alteration in the vessels of the kidney itself, the circulatory changes in the arterially aged man, when widespread, are in themselves competent to produce a state of increased renal irritability, as it were, and to further the action of a congeries of renal noxæ, without of themselves inducing a permanently recognizable change in the urinary habit or constitution. The spectral "albumin, a trace," is a very probable visitor of the man with palpable radials, but his calls may in some instances be very often repeated before he settles down in permanency as an unwelcome *memento mori*.

CASE I.—Mrs. G., aged sixty-five years, an active woman, rather neurotic, with nothing in her previous history that bears on the present subject. In January and February, 1904, the patient suffered from influenza, bronchial symptoms predominating, and was convalescing in bed when I first saw her. She was weak and somewhat anæmic in appearance, but well-nourished and apparently in fair general condition. At no time had the urine been abnormal in amount or quality.

About the middle of February Mrs. G. began to complain of general headache, the pain becoming progressively worse and being associated with some mental dulness. The bowels were constipated; the urine somewhat diminished in amount. After a week of this condition the headache became quite severe, and one morning there was a definite clonic convulsion, more pronounced on the left side, involving the head and neck as well as the legs. Soon afterward I was called to see the patient. She was dull and stupid, breathing irregularly with partial Cheyne-Stokes respiration, though not at all comatose. The radial arteries were just palpable. Heart normal as regards position and sounds, except for definite accentuation of the aortic second. The reflexes were not exaggerated

when the patient was quiet. While observing her, two minor convulsions were seen, affecting mainly the left arm and the face. The pulse was not rapid, regular, and of a deceptive and peculiar character that has been dealt with in another place[1]—so small as to suggest "weakness" (low tension) to the palpating finger. Acting on the apparent indication of the touch character of the pulse, strychnine (gr. $\frac{1}{20}$) had been given hypodermically, but only with the effect of exaggerating the spastic symptoms as well as the subjective malaise. There was very slight œdema of the ankles. A measurement of the systolic blood pressure was made at once and it was found to be 235+ mm. Hg. (Riva-Rocci, narrow arm-piece, Cook's old design). The absence of inequality of the pupils, of any palsy, of deviation of the head or eyes, and the state of the reflexes seemed to negative the possibility of an intracranial lesion, and the urine was at once examined. It was pale and clear, of specific gravity 1005, acid in reaction. There was a large trace of albumin, no sugar, no indican, urea only 6 gm. to 1 litre. There were a few casts, both hyaline and granular, but no abnormal cellular elements nor blood.

Liquid diet, purgation, diuresis, and vasodepressants were advised, with intermission of all vasostimulant medication. Within twenty-four hours the blood pressure had fallen below 200 mm. Hg., and in thirty-six hours to 108 mm. Hg. The urine still contained albumin and casts as before. Headache ceased. In forty-eight hours the urine was free from casts, it contained the faintest determinable trace of albumin, and its specific gravity had risen to 1018, while the systolic blood pressure was 165 mm. Hg. There was no suggestion of return of the convulsions, and the patient's subjective well-being was quite restored by the fourth or fifth day, when the blood pressure oscillated between 130 and 155 mm. Hg., and the urine was free of albumin and casts, of specific gravity 1020, daily quantity 1800 to 2100 c.c., urea 20 to 24 grams in the twenty-four hours.

In the past year there has been no return of symptoms. Frequent urinary examinations consistently fail to reveal any trace of a pathological condition.

In this case we have condensed into the space of a few days the major features and events of chronic (arteriosclerotic) interstitial nephritis. The absence of blood in the urine, of suppression, of every characteristic point, prevent the ascription of the term "acute inflammatory" to the renal process, while the headache, the convulsions, the slight œdema, every general symptom and sign, debar us from describing the urinary changes as those of a "grippal," toxic or febrile, insignificant albuminuria, and yet for over a year since the attack recited the patient has passed urine of unimpeachable quality and quantity,

[1] Cook and Briggs, loc. cit., p. 500.

and has been quite free of any symptoms that might point to the great twin filters. Aside from the accentuation of the aortic second sound, which persists, and the slight recognizable sclerotic changes in the peripheral vessels, physical examination today reveals nothing abnormal. The blood pressure is constantly below 170 mm. Hg. Mrs. G.'s sole complaint is of a tendency to precordial fluttering sensations, without dyspnœa, on exertion—and these appear to be perfectly controlled by small doses of the iodide. That her regimen of "much fluid, little meat," and avoidance of overstrain, together with an occasional course of the great alterative, has preserved her, for the present at least, from permanent and serious renal damage, seems a just assumption.

CASE II.—Mr. S., aged fifty-eight years, consulted me in June, 1904, for headache, anorexia, diarrhœa, and slight vertigo, which had troubled him for two or three days past. No previous attack of this nature. No infection for many years previously. Smokes a great deal, is a moderate but regular user of alcohol, works hard, and lives well. Does not rise at night to urinate. Is never dyspnœic and never noticed any œdema of ankles. Is of a costive habit.

On examination the patient shows a marked degree of general arteriosclerosis. The cardiac apex is in the fifth interspace, 11 cm. from the midsternal line. Heart impulse forcible. The aortic second sound is ringing and accentuated; otherwise the sounds are normal. Pulse regular, 68 per minute, very hard. Systolic blood pressure 235 mm. Hg.[1] in the recumbent posture. Urine, acid; specific gravity, 1018; a trace of albumin; a few hyaline casts, but no other formed elements; urea (the next day) 24 gm. in twenty-four hours, with a total quantity of 1850 c.c.

Treatment. Alcohol and tobacco proscribed for the time being. Purgation. Sodium nitrite gr. ii t. i. d. for three days; then potassium iodide gr. viii t. i. d. Meat once a day. Moderate open-air exercise.

By the fourth day the patient's blood pressure had fallen to 175 mm. Hg. (sitting posture); he was quite free from symptoms, and the urine of specific gravity 1022, twenty-four hours' quantity, 1900 c.c., showed no albumin or casts. A week later the urinary findings were the same. Blood pressure (sitting) 160 mm. Hg.

Twice since then has Mr. S. been afflicted by precisely similar symptoms, once a few days after an excessive meal with subsequent constipation, and once in association with exposure and slight alcoholic excess. In the second attack there was for two or three days a little œdema about the ankles. On each occasion albumin and

[1] Though the figures presented in this paper, having been obtained with a narrow (5 cm.) armlet, are *not* absolute, and so subject to criticism, it is to be remembered that what interests us here is the change in the blood pressure, not its absolute height. The figures obtained with a 12 cm armlet would differ from those here presented; but the facts and our conclusions would not be altered. (Janeway, op cit., p. 81.)

casts appeared in the urine, in association with blood pressures of from 200 to 250 mm. Hg., and the reduction of hypertension to 180 mm. Hg. or below was in each instance readily accomplished and followed by a rapid disappearance of abnormal constituents from the urine. Repeated examinations of the patient in the intervals, when he is always subjectively in splendid condition, show blood pressures constantly between 165 and 185 mm. Hg., and urine absolutely normal as regards quantity, specific gravity, albumin, casts, and urea.

The case is striking. That albuminuria and suburæmic symptoms should have appeared three times within nine months, and leave no perceptible lasting injury behind them, must speak well for the quality of renal parenchyma provided in Mr. S.'s construction. Knowing the patient's habits and disposition, it seems almost certain that the next few years will deal more severely with him, and that at no distant date his urine will show a constant trace of albumin, and that his intervals of complete well-being will be at the least clouded.

CASE III.—Mr. W., aged eighty-two years, a fairly well-preserved man of his years, with advanced sclerosis of all the peripheral arteries. Has been operated upon for prostatic enlargement, and has constantly some pyuria, but without any evidences of nephritis. Blood pressure is constantly 180 to 200 mm. Hg. (arm 23 cm. in circumference).

Between August, 1904, and January, 1905, Mr. W. had five convulsive seizures. Three of these I have seen. In each instance the patient, after feeling vaguely unwell for a period varying from ten minutes to as many hours, has complained of peculiar sensations in the right hand and arm, soon followed by definite clonic spasms of the extremity. The attacks have lasted for from two hours to twelve, have always involved the head and neck on the same side, and on three occasions the right leg has been involved in the rhythmic spasm, which is quite suggestive of that of the second stage of epilepsy—the movements are rapid, 120 to 160 to the minute, vibratory, and in violence quite exceed that of any tremor. In their distribution and character they have been strikingly suggestive of the unilateral posthemiplegic movements described by Weir Mitchell. Two attacks which I witnessed were associated with marked, but transient, motor aphasia. With the cessation of the movements Mr. W. is left in absolutely unchanged condition, except for an exaggeration of the tendon reflexes on the right side, which is never apparent after two or three days. The attacks are evidently merely a cerebral symptom of Mr. W.'s extensive arteriosclerosis, transient attacks of a hemiplegic nature, from which, as Osler recognizes, the recovery tends to be perfect. That they are, moreover, determined in their onset by a vasomotor element, seems to be evident from the blood-pressure observations which I have been able to make. Mr.

W.'s blood pressure in three attacks observed has been between 230 and 300 mm. Hg. At no time in the intervals between attacks has it ever been found above 200 mm. Hg. Blood pressure lowering medication (sodium nitrite) has always been efficient in terminating the attack, and the patient has had no further seizures since the institution of a regimen of potassium iodide, with an occasional course of the nitrites when indicated by the blood pressure.

Of interest in connection with the preceding cases is the fact that urine passed during two of the attacks personally seen contained large numbers of hyaline and granular casts, while the urine of the intervals only contains cylindroids, with at most an occasional hyaline cast. At the end of one seizure the urine (specific gravity 1018) contained a trace of a reducing substance which yielded CO_2 on fermentation with yeast. The trace of albumin constantly present is no more than may be referred to the amount of pus in the urine.

CASE IV.—C. M., laborer, aged thirty-nine years. No lues; moderate with alcohol; no infections since those of childhood. The patient's father died of apoplexy at forty-eight, his mother of Bright's disease, and an older brother has kidney trouble. This man complained of little but vague digestive and other apparently neurotic symptoms, but his radial arteries were easily palpable, his systolic blood pressure was 190+ mm. Hg., his heart slightly enlarged, and the aortic second sound was accented, while his urine contained a considerable trace of albumin and some cylindroids, but no casts. Reduction of the blood pressure to an average of 160 to 170 mm. Hg. by measures of personal hygiene, regulation of the bowels, the nitrites, and small doses of potassium iodide, was attended by substantial betterment of the subjective condition, and the albuminuria became inconstant, instead of being evident in every specimen, as when first seen. What is of interest is that when further attempts at vasodilatation were made, and the blood pressure was reduced to a mean level of 150 mm. Hg. or below, the albumin appeared in much larger traces and constantly, and the patient complained of feeling as badly as when his blood pressure was allowed to remain around the 200 mm. Hg level.

Further critical discussion seems unnecessary, and the histories cited, to those familiar with the writings of Cook, Janeway, Clifford, Allbutt, and others, may in themselves justify the following statements:

1. Albuminuria (and cylindruria) in the subjects of generalized arteriosclerosis, where no other cause is at work, is always accompanied by a state of high blood pressure.

2. Intermittent attacks of hypertension of the pulse and concomitant albuminuria, separated by longer or shorter intervals of normal blood pressure during which the urine is normal, may be the only clinical expressions of a generalized arteriosclerosis.

3. Therapeutic relief of hypertension in this general class of cases, if not carried too far, will tend to relieve the pathological condition of the urine. In a certain number of cases we may reasonably hope by this means to postpone the onset of chronic renal changes.

TINEA VERSICOLOR IN AN INSTITUTION.

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Although it is one of the mildest of parasitic diseases and, in fact, hardly more than an epiphenomenon in the struggle of the organism with disease, tinea versicolor has, nevertheless, had an unusually interesting history, and one which strikingly illustrates the ups and downs of scientific opinion. In spite of two fairly clear differentiations, the earlier writers persisted in confusing the lesions with pigmentary diseases such as vitiligo, chloasma, and lentigo. After the discovery of the fungus pathologists attempted to identify it with tubercle bacillus, with non-pathogenic organisms, and with the fungi of other skin diseases. Clinicians have long vainly sought to prove that the affection is frequently associated with certain conditions of the skin and certain constitutional diseases. And systematic dermatologists have dogmatically held that the disease is confined to certain parts of the body and to certain periods of life.

In the support and dispute of these and various other opinions the subject has been considerably exploited and many points have been settled. Considerable uncertainty still exists, however, in regard to the classification of the parasite and the means of cultivation, but more particularly, from a clinical standpoint, in regard to the predisposing causes of infection, the question of contagion, and the age limits. The following observations were made with a view of obtaining, if possible, some information on these points, and were suggested by an opportunity of observing a series of cases at an institution which seemed well suited for such a purpose and by the possibility of consulting the literature upon the subject in general.

Clinical History. As Sennert says, it is incredible that the disease did not exist among the ancients, but no information concerning its antiquity is obtainable because, as he explains, we do not know by what names many kinds of macules and tubercles were designated by the Greeks and Romans. The earliest reference found is that of Solenander, who in 1596 described the affection, without naming it, as a symptomatic one, arising

"ex succo et humore melancholico," and prescribed vegetable drugs. Sennert in 1629 wrote a section, "De maculis a Germanis epaticis dictis," and says that the lesions and the German name "Leberflecke" are very well known to the laity. He holds that the cause is undoubtedly a melancholy humor, but announces distinctly that the spots are different from the lentigines. In 1799 Robert Willan introduced the term pityriasis versicolor as synonymous with maculæ hepaticæ, and says that probably chloasma and vitiligo should not be included under this head. He notes that the spots have been attributed to eating fruit, mushrooms, etc.

The statements of Sennert and Willan on the classification of the lesions stand out from the confusion caused by the majority of early writers who ranked them either with lentigo (Platerus), vitiligo (Sauvages, 1768), ephelis (Alibert, 1814), or chloasma (Wilson, 1863). Even after the discovery of the fungus in 1846, the uncertainty remained nearly as great through the efforts of the clinicians, to say nothing of those of the pathologists. Thus, Wilson denied the existence of the fungus, and Bazin attributed all pigmentary lesions to it. Jonathan Hutchinson, who in 1859 thought that a case of pityriasis versicolor had been contracted from a case of ringworm, as late as 1889 held that pityriasis versicolor, ringworm, and eczema marginatum were all due to one and the same fungus. In the course of time, however, it has become gradually and generally recognized that the disease is due to a specific organism.

When we turn to other aspects of the question, we find a conflict between various clinical dogmas and an appeal to cases. One of the oldest and most firmly fixed dogmas was that the lesions occur only on parts of the body covered by clothing. For instance, Kaposi stated that the face, hands, and feet are never affected. Another belief much in vogue relates to the age limit; thus Kaposi again says the disease is never met with in children or in the old. And almost every writer has something to say on predisposing causes, such as oiliness, dryness of the skin, excess of perspiration, or intestinal and general constitutional diseases.

These views have been contested by reports of cases in point. In 1880 Unna cited the case of a bearded man, the lower part of whose face was affected, and quoted his own case to the same effect. In 1885 Biart reported a case in which the lesion had extended from the neck over the left cheek and onto the forehead. In 1896 Smith, in answer to a statement in Morrow's *System*, told of a man the dorsal surfaces of whose feet were alone affected. In 1899 Gottheil gave an account of a Cuban physician who had had black spots on only his left palm for fifteen years. These proved to be due to the organism in question. In 1899 Powell was prompted by a dictum of Bosanquet to note that the face is frequently affected in Assam—a fact which he attributes to lack

of soap and frequent bathing. In 1901 Allen referred to a number of cases of versicolor of the face, and gave photographs of three cases. Thus the face, hands, and feet, though infrequently infected, are shown to have no immunity.

In regard to age, data are scarce. In 1887 Phillips exhibited a boy of seven years who had symmetrical lesions on the back and chest; and Matakieff, in 1899, found an infection in 10.8 per cent. among over 1000 old patients.

As to predisposition, Hublé, in 1886, denied the influence of any special season, mode of life, sex, nutrition, or temperament, and says we must acknowledge our ignorance, unless we are willing to fall back on "idiosyncrasy." Allen, in 1901, failed to find the infection associated with phthisis, dyspepsia, seborrhœa, or any severe disease.

A few miscellaneous features have been reported. Taylor, in 1873, related the case of a man who had been on large doses of potassium iodide for four days, when his entire back became bluish-black. It was found that the patient had tinea versicolor of the back, had worn a freshly starched shirt, and the coloration of the reaction between the iodide and the starch had been taken up by the scales. Unna has described several cases of a rapidly growing ring-formed lesion. Allen, in 1901, found an infection under the hair of the pubis frequently associated with infection of other parts of the body, and believes that this is a source of reinfection. He also speaks of Lugol's solution or the tincture of iodine to distinguish parasitic lesions, which turn a mahogany color, from non-parasitic lesions. Sobel, a few months later, wrote an article on "Pityriasis Versicolor, with Special Reference to Allen's Iodine Test," in which he testified to its value. It is undoubtedly of some use, but was introduced in 1882 by Besnier and Balzer.

History of the Parasite (*Microsporon furfur; Malezzia furfur*). The term microsporon was introduced into the literature by Gruby, the eccentric physician and investigator of Paris, who in describing the fungus of phytoalopecia in 1843 called it microsporum (1844, microsporon), on account of the smallness of the spores.

In 1846 Eichstedt, of Griefswalde, announced the discovery of the parasite in a short paper, "Pilzbildung in der Pityriasis Versicolor," including two minute drawings, but gave the fungus no name. His attention had been directed to the subject by a visit from a laborer with an extensive skin affection, who said that he had contracted the disease from sleeping with another laborer, and that in turn he himself had passed it on to his brother in the same way. Eichstedt made a scraping of the skin and found the spores, and, after clearing with caustic ammonia, found the threads as well. After observing the same elements in other cases, he became convinced that pityriasis versicolor, as well as prurigo

lupinosa (favus) and aphtha, was produced by a fungus, and expressed the hope that this discovery would put an end to the internal treatment of the disease.

In 1853 Charles Robin, in a systematic work, made a genus out of Gruby's microsporon, and for Eichstedt's organism made the species microsporon furfur. He said he had not verified the facts himself, but from Eichstedt's and Sluyter's descriptions and drawings he concluded that the organism was generically a microsporon, and had the specific differences from the other forms already described.

The discovery of fungus was soon followed by attempts at pure culture, inoculation, and classification; but while inoculation has been accomplished, the question of culture and classification is still unanswered. Kobner in 1864 reported that after a long time he succeeded in infecting himself over the sternum and then transferred the growth to rabbits. Grawitz in 1877 had obtained cultures which he believed showed that the organisms of favus, tinea tonsurans, and pityriasis versicolor were identical with oidium lactis, but in 1886, after some further work, became convinced that they were all independent. Neuman in 1880 said that he had observed under the microscope a segmentation of the spores and the production of tubes from spores.

In 1886 Duguet and Hericourt seemed to be on the point of making a new epoch. They cited three cases of consumption in which no bacilli were found, but in which occurred elements resembling those of microsporon furfur. These, when injected into animals, produced tubercles; when grown in milk or bouillon, bacilli appeared at the top of the tube, while lower down were found the anaërobic spores. By a change in the reaction of the media, or by heat or cold, they made the spores replace the bacilli. They concluded that the bacillus of Koch was but one stage of the growth of microsporon furfur, and believed that this discovery would explain why the bacilli are not found in tuberculous glands, etc., and would affect prognosis and treatment. Nothing has since been heard from these gentlemen, and their experiments have been repeated and results disproven, although of course they were never to any extent accepted. It is improbable that they were dealing with either tubercle bacilli or with microsporon furfur.

Hublé in 1886 wrote an article to prove that the disease is transmissible, especially by sexual intercourse, and expressed the belief that this fact would be of medicolegal importance in some cases of assault. He also gave instances of indirect transmission by infected clothing, and succeeded in infecting himself and a companion in the fold of the elbow by applying the scales in glycerin; the growth appeared in two to six weeks. Lancereaux's account in 1875 of the accidental infection of himself and wife had been considered inconclusive by Besnier and Balzer in 1882.

Many names have been proposed for the parasite, but, aside from microsporon furfur, the only one of importance is Malezzia furfur, which Baillon proposed in 1889 in honor of Malezzia. While he did not give any valid reason for rejecting the term microsporon, he believed that the differences were great enough to justify a new name.

Sehlen in 1889 had obtained likely cultures, but had no success at infection; and in 1890 Sehlen and Unna obtained further cultures in 5 out of 7 cases, but were unable to infect anyone with them.

Kotliar in 1892 claimed to have obtained a pure culture, and to have infected rabbits with it; but its cultural characteristics threw doubt on its specificity. Spietschka in 1896 reported that after many failures on various kinds of media he had obtained pure cultures from 12 cases, had infected man, and had reobtained the parasite in pure cultures—thus fulfilling Koch's postulates for a specific organism.

Vuillemin in 1899 justified Baillon's term Malezzia furfur on the ground that he had discovered a new specific character, namely, spiral ridges on all globules, including those formed by budding of the other globules, those springing from the mycelia, and those which give rise to mycelia. He said that until the discovery of some such character it was questionable whether the filaments and globules belonged to the same organism. Matakieff, a pupil of Vuillemin, in a thorough-going thesis in 1899, recorded the presence of the ridges in a large number of cases. He also found the bacillus mesentericus fuscus present in the brown scales, but not in the lighter scales, and concluded that it has something to do with the coloration of the lesions.

Sabourand in 1900 expressed a doubt that any author had surely obtained a pure culture on artificial media. This sort of remark prompted Matzenauer in 1901 to state that Spietschka had settled the question, and to give an account of a pure culture he had obtained two years before. Out of several hundred plates he isolated two pure cultures, but was unable to infect the skin with them.

In regard to classification, Pinoy, Goedelst, Vuillemin, and Sabourand agree that position of the organism is undetermined. Pinoy favors putting it among the fungi imperfecti along with streptothrix, while Vuillemin inclines to the ascomycetes, on account of its modes of reproduction .

Special Observations. The cases observed occurred among 709 male patients in the service of Dr. H. R. Hummer at the Detached Buildings in the fall of 1904. As far as could be learned none of the patients had previously been treated for this disease. All the patients were examined stripped, and 113 were found to present lesions which might possibly be those of tinea versicolor. These cases were then taken up in groups and scrapings made

in each case. The scales were mounted in equal parts of liquor potassæ and glycerin and examined after a few hours with the high power; if necessary, fat and foreign coloring matter were removed by alcohol and ether, and several specimens were examined.

Forty-two cases, or 5.92 per cent. of the population, proved to be infected. Of the 71 negative cases about one-third were instances of seborrhœal eczema; another third resembled a special group of infected cases which will be mentioned later, and the remainder were divided between the desquamation of the last stages of tuberculosis, senile atrophy, and a few undetermined cases.

In regard to frequency, the number is fairly high. Hublé, of Toulouse, found an infection of 1.6 per cent. among 2360 soldiers between the ages of nineteen and twenty-four. In a part of India 4 per cent. of 2540 prisoners were infected (Bulkley). Matakieff reports an infection of 6.25 per cent. among 80 patients at a hospital in Nancy, and the infection of 10.8 per cent. among 1201 old patients at another hospital. In an apparent epidemic of the infection in Hamburg during 1879 Unna found 20 per cent. of 82 prostitutes infected. In the Long Island State Hospital for the Insane Winfield found an infection of 1.29 per cent. of 1084 patients. No reason is apparent for this small figure in a similar institution; it may be stated, however, that two-thirds of the subjects examined by Winfield were females who are less susceptible according to some statistics, but more so according to others.

In analyzing the cases attention was paid to any factors which might be operative, such as previous occupation, length of residence in a hospital, age, organic and mental diseases, and peculiar characteristics. The most noteworthy results were obtained in regard to age and in regard to a special group of cases which presented unusual clinical and microscopic characteristics.

The following table shows the composition of the service by previous occupation, with numbers and percentage infected:

	Admitted from		Navy.			Army.		
	Civil life	Soldiers' Homes	Up to 1898.	Since 1898	Total.	Up to 1898	Since 1898.	Total.
Number examined	199	250	27	26	53	141	66	207
Number infected	16	13	0	0	0	8	5	13
Percentage infected	8 04	6 28	0	0	0	5 53	7 57	6 23

According to these results civilians who are in large part segregated are infected to a somewhat greater degree than men from military life. Men from the navy are singularly free from the parasite. In spite of their age, inmates from the Soldiers' Homes show a considerable infection. The increased percentage in men from the army since 1898 is not due directly to tropical service, as only one out of a number of soldiers who had been in Cuba or the Philippines was infected upon admission.

The following table shows the frequency of infection according to length of residence in the hospital:

Years of residence	—1	1-3	4-10	10-20	20	Total.
Number examined	68	81	254	139	167	709
Number infected	5	3	16	7	11	42
Percentage infected	7.35	3.69	6.29	5.03	6.58	5.92

During the time in which records of careful physical examinations have been kept 8 of the cases have been admitted; 2 belonged to a special group which might easily have escaped attention, 2 were infected upon admission, and 4 have been infected since admission. No especially infected ward was found, and the general condition of contagion of skin diseases may be understood from the fact that a single case of pediculosis lived in a ward with 50 other men for a number of weeks without infecting any of them. Patients are bathed every week and given sterilized underclothes, so that there is no special reason for infection; still the fact remains that it occurs.

The following table shows the distribution of the infection by age of the patients:

Age	—29	30-39	40-49	50-59	60-69	70-79	80	Average.
Number examined	59	96	108	144	193	99	10	51 yrs.
Number infected	3	5	7	8	12	7	0	57 "
Percentage infected	5.08	5.22	6.48	5.55	6.21	7.77	0	
Special group:								
Number infected			3	4	4	4	...	60 "
Percentage infected			2.77	2.77	2 07	4.01		

Hence, the largest number of infections occur among the old patients. The average age is fifty-seven. Bulkley states that tinea versicolor is not common over fifty; but of this series two-thirds of the cases were over fifty, the oldest was seventy-seven, the youngest twenty-six.

Fifteen patients, or 35 per cent. of the whole number, formed a special group. These were, as a rule, elderly men with abundant sternal hair, in which could be found coarse white or yellowish scales in distinction to the clear skin and fine, branny scales usually found. Microscopic examination of these scales did not show the usual mycelia, but only the spores. In some cases these were grouped in small numbers as usual, but in others they occurred lying together in almost solid sheets. There was no doubt in regard to their presence, although it was impossible to determine with any certainty the ridges mentioned by Vuillemin either in these spores or in those accompanied by mycelia. The average age of this group was 60.8 years, oldest seventy-seven, youngest forty-one. Some 20 other patients of similar age showed the same coarse scales, but no spores could be found in them. It seems probable that as in old cultures the spores alone are said to be

produced, so in old age the same resting stage may be reached, and in some cases the spores themselves may disappear.

In regard to organic disease, only one case was found among some 30 consumptives, and this patient was practically cured. However, a number of cases were found with diffused scaling which might have been mistaken for tinea versicolor except for careful microscopic examination. After looking for associated oiliness and dryness of the skin, or digestive disturbance, etc., the conclusion was reached that no connection could be demonstrated in these cases.

The question of possible association of the infection with nervous and mental disorders would naturally deserve special consideration; and in view of the number of systemic changes which are thought to be correlated with various neuroses and psychoses, and the number of theories already mentioned, it was not unlikely that at least some hypothesis might be reached. None was, however. The classification under which most of the patients were admitted has become obsolete, and many of the patients have become more demented. While the cases could be classified, as Winfield's were, into so many chronic manias, chronic melancholias, etc., they would signify nothing. The most that can be said is that the disease is fairly frequent among the chronic insane; that, incidentally, 7.5 per cent. of 80 epileptics were infected. 43.8 per cent. of the cases showed infection of the pubic hair, which might easily have been overlooked. No evidence of systemic reaction was found in the blood; 11 of the cases were extensive, and the most extensive showed a normal condition with 9800 leukocytes and 1.5 per cent. of eosinophiles. A similarly extensive case of tinea circinata gave a leukocyte count of 7800 and 2 per cent. of eosinophiles.

Dr. Cornelius de Weese, while at the Hospital, made several attempts at cultivation, and succeeded in isolating a fungus which liquefied gelatin and showed other so-called characteristic properties; but its variable morphological characters have so far made any conclusions impossible.

In the way of treatment, various antiseptics have been used, but none has proved as rapid and effective in action as sulphurous acid developed by Crocker's method of first applying to the scales sodium hyposulphite, ℥j to ℥ij, and then adding tartaric acid, ℥ss to ℥ij.

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THE CLINICAL, BACTERIOLOGICAL, AND METABOLIC ASPECTS OF A CASE OF TRAUMATIC TETANUS; TREATMENT WITH TETANUS ANTITOXIN; RECOVERY.[1]

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I. CLINICAL COURSE AND TREATMENT.

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INASMUCH as the reported cases of true traumatic tetanus treated with subcutaneous and intraspinal injections of antitetanic serum

[1] Read before the Harlem Medical Association, N. Y., May 3, 1905.

are not numerous, we feel justified in placing on record the facts of the following case:

The patient, a boy twelve years of age, was admitted to Mount Sinai Hospital on the night of August 11, 1904. Two weeks previously he had stepped on a nail, which perforated the sole of the shoe and penetrated the ball of the right foot. A physician who was summoned incised the wound, which, ten days later, was entirely healed. There were no constitutional symptoms till eleven days after the accident, or three days before admission. Early on the morning of that day, without warning, the boy fell to the floor in his home. There were no convulsions or unconsciousness. Saliva dribbled from the mouth and the boy complained of pain in the back. After that he was apparently sick; he complained of severe backache, loss of appetite, and on the morning of the day of admission he was unable to open his mouth. He was still able, however, to swallow fluids. At about 11 A.M. there were noticed a staring countenance, rigidity of the neck, back, and legs, and hyperacusis. There were noticed, also, twitching of the muscles of the face, trunk, and extremities. There were, according to the family, no headache, unconsciousness, fever, chills, or involuntary urination. The bowels had not moved in three days. Perspiration was very profuse. The chief complaints were inability to open the mouth and pain in the back.

On admission a *physical examination* by Dr. Bauman, the house physician, showed the following: General condition fair. There are present trismus, risus sardonicus, opisthotonos, rigidity of the neck, and contraction of the muscles of the abdomen and back. No facial or ocular palsy. The pupils are equally contracted. The throat could not be inspected. No difficulty in swallowing fluids. Slight enlargement of the lymph nodes in the neck, axillæ, and groins. Kernig's sign is present. The Babinski reflex is present. The patellar reflexes are exaggerated; heart and lungs negative; liver palpable two fingers below the free costal border; spleen negative; abdomen somewhat rigid; temperature, 99°; pulse, 104 in frequency, of good quality; respirations, 18; they are irregular, and at times there is stridulous inspiration. The urine shows a trace of albumin, otherwise negative. On the sole of the right foot, in the region of the distal extremity of the first metatarsal bone, is a small scab about the size of a lentil. The house physician immediately removed the scab; under aseptic precautions he incised and curetted the wound, and then cauterized it with silver nitrate. In the scrapings was a small, black, foreign body resembling leather in appearance; this, together with the curetted tissue, was sent to the pathological laboratory of the hospital, a report from which is included in this paper.

On August 11th, the night of admission, there was given in the right pectoral region a subcutaneous injection of 20 c.c. of Board of

Health tetanus antitoxin. An hour later a lumbar puncture was performed and 5 c.c. of cerebrospinal fluid were withdrawn. This was followed by a subdural injection of 20 c.c. of tetanus antitoxin. During these procedures the slightest sound or touch would cause the boy to start. In attempting to swallow some fluid nourishment, the muscles of his entire body suddenly became contracted. Twelve hours after the first subcutaneous injection another one was given. The wound was dressed and touched with a 10 per cent. solution of nitrate of silver.

August 13*th.* Under chloroform narcosis, after withdrawing 25 c.c of cerebrospinal fluid, 20 c.c. of tetanus antitoxin were injected subdurally. Six hours later 20 c.c. of tetanus antitoxin were injected subcutaneously into the thigh. A leukocyte count made on this day was 12,600.

14*th.* During the three days of observation there has been little change in the general condition. The mental condition has been clear. Fever but slight. The spastic signs persist; there are still present trismus, risus sardonicus, opisthotonos, and hyperacusis. While attempts to raise the patient's head are followed by contraction of the muscles of the neck and back, lateral rotation of the head can be performed with ease. The patient swallows and retains sufficient fluid nourishment. The medication has been chloral and bromides, at first per rectum, later by mouth, and whiskey per mouth. The intraspinal and subcutaneous injections of antitoxin were given to-day as previously.

16*th.* Subcutaneous injection repeated. Catheterization was necessary yesterday.

17*th,* 18*th,* and 19*th.* Spastic signs persist. A subcutaneous injection of 20 c.c. of antitoxin given on each day.

20*th.* The temperature has risen to 102° and an erythematous eruption has appeared on the chest, abdomen, back, and thighs. White blood corpuscles, 13,800. Trismus, rigidity of the neck, and opisthotonos still present. General condition unchanged.

26*th.* The erythema, fading on the trunk, is now present on the face. No change in the general condition or spastic signs. White blood corpuscles, 10,400.

28*th.* The general condition is improving, the spasticity is diminishing. The temperature is still betwen 100° and 102°; rash almost disappeared. Examination of the heart and lungs is negative. The urine, except on the day of admission, has been negative.

September 1*st.* Continuous improvement in the general condition. White blood corpuscles, 7800. The rash has faded. The temperature is normal.

7*th.* The general condition is good. The spastic signs have disappeared.

16*th.* Patient discharged cured. No symptoms remain.

While a study of the clinical features of this case shows that we

are dealing with a true case of traumatic tetanus, it is worthy of note that an attempt to inoculate ordinarily susceptible animals (white mice and guinea-pigs) with the tetanus bacillus found in the scrapings and foreign body from the wound was not successful.

In the course of treatment between August 11th and 19th, there were given three intraspinal injections of 20 c.c. each, and eight subcutaneous injections of 20 c.c. each, the Board of Health antitoxin being used. To attribute the recovery in this case directly and solely to the antitoxin injections would perhaps be going too far. We cannot help asking ourselves the question: Would a patient with equal resisting powers contracting the disease in the same way have recovered without the antitoxin treatment? We can only say that, in view of the almost uniformly fatal termination of the disease, it is not unlikely that the antitoxin treatment probably effected a cure in this case. When we remember the channel through which the tetanus toxins reach the central nervous system, and when we recall the experiments of Wassermann showing that the union between tetanus toxins and their antitoxins is one produced by a chemical affinity, it would appear that both from a therapeutic and prophylactic standpoint the intraspinal injections are preferable to the subcutaneous or cerebral injections, for by the former method the antitoxin comes in direct contact with the toxins of the disease. This case was taken from the ward of Dr. Koplik during my service.

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II. Bacteriological Examination.

by leo buerger, m.d.

The material from which cultures were made consisted of a small, dirty, black, foreign body, apparently a piece of leather, the fatty tissue immediately surrounding it, and scrapings from the wall

of the cavity in which the foreign body was situated. The latter was located at the upper end of the channel through which it had been forced by the traumatism, and was embedded in discolored, necrotic, but non-purulent fat and granulation tissue. The tissue in the immediate neighborhood of the apparently healed external wound was not employed, the examination being confined to material obtained from the depth.

In spreads made from the tissue there were a few leukocytes, red blood cells, and Gram-positive diplococci which were regarded as probably being staphylococci. No organisms resembling tetanus bacilli nor any spores could be found.

The method which Kitasato has recommended for the isolation of the tetanus bacillus was used. This depends upon the observation that its spores can resist exposure to high temperatures for considerable periods of time. Three series of cultures were made: one from the foreign body, another from the tissue in its vicinity, and a third from the cerebrospinal fluid evacuated before the first antitoxin injection. The mode of procedure was the same in each instance. At the end of twenty-four hours' incubation the usual methods of isolation showed the presence of staphylococcus albus and bacillus coli communis in the cultures taken from the foreign body, and staphylococcus albus alone in the tissue. The cerebrospinal fluid remained sterile.

All the cultures were then exposed to a temperature of 80° C. over a water-bath for forty minutes on two successive days. This procedure was followed by incubation under strictly anaërobic conditions (in a hydrogen atmosphere) of both the original tubes and subcultures. Thus pure cultures of tetanus bacilli were obtained from the foreign body and from the subcutaneous areolar tissue.

Although the bacillus isolated in this case resembles the typical tetanus bacillus in almost all of its cultural and morphological characteristics, it presents certain slight variations which are deserving of mention. Firstly, it shows a marked tendency to the formation of oval rather than spherical spores. Secondly, there is perhaps more than the usual amount of variability in regard to the Gram procedure. Whereas it stains positive for the most part, even young cultures are apt to show many bacilli which either partly or wholly decolorize.

On culture media it differs little from the ordinary type. It coagulates milk, and its colonies in gelatin and on agar show less tendency to filiform or lace-like outgrowths.

Animal Experimentations. An interesting feature presented by this bacillus is the absence of virulence for the animals ordinarily susceptible. Whereas white mice and guinea-pigs succumb rapidly to subcutaneous inoculation with the tetanus bacillus, I was unable to kill or produce tetanic symptoms in either guinea-pigs or white mice by the injection of cultures of this bacillus. The following

is a brief *résumé* of the experiments on the pathogenicity of the bacillus.

One guinea-pig and one mouse were inoculated directly with some of the material from which the cultures had been made, and another mouse with the cerebrospinal fluid.

Guinea-pig No. 1. The foreign body (piece of leather) was introduced into the abdominal wall through a small incision, and the wound was closed with silk sutures. Result: no symptoms; in three weeks the foreign body was expelled and spontaneous healing took place.

Mouse 1. Three cubic centimetres of sterile normal saline were thoroughly mixed with some of the tissue about the foreign body. The fluid was injected into the right groin. Result: negative.

Mouse 2. Two cubic centimetres of cerebrospinal fluid were injected into the right leg. Result: negative.

From these experiments the inference is clearly to be drawn that the material from which the bacillus was isolated could not have carried with it tetanus bacilli or spores virulent for these animals. All three animals survived and in none of them were any tetanic symptoms discernible.

Another series of experiments for the purpose of ascertaining the virulence of the bacillus in cultures was made. Four guinea-pigs were inoculated in the leg with varying quantities of bouillon cultures, the smallest dose being one loopful of a four-day growth, the largest 2.5 c.c. of a seven-day culture in plain broth. Three white mice were similarly treated, the largest dose being 1 c.c. of a four-day culture. There was no reaction in any of these animals. With a view to determining whether predisposing factors such as the association of other bacteria or local traumatism would aid in the production of tetanus, another series of animals was employed. Neither by the addition of the bacillus coli communis, nor by the introduction of wood splinters impregnated with the spores of the organism, nor by local traumatism, such as fracture of the bones of the leg, will I be able to give any positive results in guinea-pigs or mice. One guinea-pig died of staphylococcus aureus sepsis, and all the others recovered.

In a recent contribution to the study of tetanus, Vincent[1] calls attention to the influence of heat in heightening the susceptibility of animals to infection with tetanus. By carefully raising the temperature of guinea-pigs in the incubator, their power of resistance was so diminished that even very small doses of only slightly virulent strains would rapidly produce tetanic convulsions. In this way he was able to produce a true tetanus septicæmia. A similar experiment with my bacillus gave negative results. A guinea-pig received an injection of 2.5 c.c. of a bouillon culture

[1] Contribution à l'étude du tétanos, etc., Annales de l'Institut de Pasteur, tome xviii.

in the back. It was then exposed to a temperature of 43° C. in an incubator. In four hours its temperature had risen to 43° C., and twenty minutes later had fallen to 42° C. It was then taken out of the incubator and put under observation. Here, again, the result was negative.

A careful consideration of all the above data naturally leads us to the question as to whether we are dealing here with a true tetanus bacillus.[1] Tavel has described an organism which greatly resembles the true tetanus bacillus, but which is non-virulent for animals. He has found it in many cases of intra-abdominal abscess and especially in appendicular affections. He regards it as an important factor in the etiology of appendicitis. Dr. Libman (personal communication) has also met with a similar organism in pure culture in a case of appendicitis. Tavel's pseudotetanus bacillus, however, although possessing oval spores, differs from the strain that I have isolated in the following points: First, its spores will not withstand a temperature of 80° C. Second, it never possesses more than from eight to twelve flagella, whereas the bacillus under consideration has been found with as many as twenty or thirty.

We must conclude, therefore, that we are in possession not of a pseudotetanus, but of a true tetanus bacillus.

The question as to whether this bacillus is to be regarded as one peculiarly pathogenic for man and avirulent for mice and guinea-pigs, or whether it should be looked upon as having lost its virulence either in the body of the patient or in the process of isolation, it is very difficult to answer. Perhaps further studies upon this and related bacteria will throw some light upon these rather vague and unsettled points. At present, with the data at my disposal, it seems most rational to classify the bacillus with the true but atypical tetanus bacilli.

III. A Contribution to the Metabolism in Tetanus.

BY EDWARD A. ARONSON, M.D.

So little has been accomplished in the metabolism in tetanus that I feel compelled to add my investigations observed in a case admitted to the wards of the Mount Sinai Hospital, in the service of Dr. Henry Heiman, during the past summer.

Before stating my results, I wish to mention that the studies were confined only to the urinary excretion—that is, a determination of the solids, both total and inorganic; the total acidity in terms of HCl; the sulphates, both preformed and ethereal; the total nitrogen; ammonia; chlorine; phosphates and uric acid. Only twenty-four-hour collections of urine were employed, and the work extended

[1] Tavel, Centralblatt f. Bact. u. P., Bd. xxiii. p. 538.

over a period of eight days, divided with an interval of five days into two series of four days each.

The methods used were as follows:

Solids: 5 c.c. of unfiltered urine in a platinum vessel were placed in an oven at 100° C. for three and one-half hours and weighed, giving total solids. HNO_3 and H_2SO_4 were added after igniting, then heated and again weighed, giving inorganic solids as sulphates.

Acidity: 10 c.c. of filtered urine titrated with phenolphthalein as an indicator and determined in terms of HCl.

Sulphates: 50 c.c. of filtered urine treated with 10 c.c. of acetic acid, heated, and 25 c.c. of barium chloride added precipitating the sulphates. This was then filtered, ignited and weighed —this gave me the preformed sulphates. To the filtrate 15 c.c. of HCl were added, boiled until decomposition was complete, filtered, and the precipitate again weighed in a platinum crucible—thus giving the ethereal sulphates.

Total nitrogen: 5 c.c. of unfiltered urine were treated by the Kjeldahl method.

Ammonia: 10 c.c. of filtered urine treated by the Schloesing method.

Chlorine: 10 c.c. of filtered urine were placed in a caserole, $NaCO_3$ and $NaNO_3$ added, and then evaporated. After igniting and taking up with hot water, HNO_3 is added to acidify, $CaCO_3$ to neutralize, and after heating on a water-bath is filtered and washed. The filtrate was then titrated with a $\frac{N}{10}$ $AgNO_3$ solution with K_2CrO_4 as an indicator.

Phosphates: 50 c.c. of filtered urine to which 5 c.c. of a solution of sodium acetate and acetic acid are added are heated and then titrated with a standard solution of uranium acetate.

Uric acid: To 200 c.c. of filtered urine Folin's method was employed.

Perhaps it would not be amiss to mention the constituents of a normal urine for purposes of comparison later on. Different authorities give different figures; hence there are no arbitrary statistics to be compared with my findings.

Folin,[1] in a very recent publication, gives as the result of an approximately complete and careful analysis of 30 "normal urines" the following figures in a general average:

Amount	143 0 c.c.	Total sulphur	3 31 c c.
Total nitrogen	16 0	Inorganic sulphate	2.92
Urea	13 9	Ethereal sulphates	0 22
Ammonia	0 85	Neutral sulphur	0.17
Kreatinin	0 58	Acidity	6.17
Uric acid	0.3386	Total phosphates	3 87
Undetermined nitrogen	0 6	Chlorine	6.1

[1] American Journal of Physiology, No. 1, vol. xiii.

These figures were obtained while the patient was subjected to the following diet:

Milk	500 c.c.	Sugar	20 gm.
Cream (18–22 % fat)	300 c.c.	Salt	6 gm.
Eggs (white and yolk)	450 gm.	Water up to	2 litres.
Horlick's malted milk	200 gm.	Water to drink	900 c.c.

In looking into the literature of metabolism in tetanus, I found a series of results in five cases, all adults, reported by Vannini,[1] and I will only mention his conclusions:

"1. The patients required considerable quantities of food, which was but poorly absorbed; hence the marked emaciation.

"2. Increase of albumin metabolism, shown by the marked increase of nitrogen excretion in convalescence.

"3. Scant, concentrated, and hyperacid urine.

"4. Urea and ammonia increased, uric acid excretion diminished.

"5. Often a slight albuminuria with casts.

"6. Rare or doubtful glycosuria.

"7. Chlorine diminished, sulphates and phosphates normal."

My case was a child eight years of age, and the table on page 276 is given with the results of the observation.

These studies were made on urine collected after the first twenty-four hours of his stay in the hospital and for the four succeeding days during which his temperature never exceeded 100.6°, but marked tetanic symptoms were present all this time. The second series of investigations were begun five days after the last of the preceding series was ended, and during the latter period the symptoms had materially decreased and the condition improved much.

In all metabolism work, one of the most important factors is the determination of the total nitrogen excretion. In accurate observations it is necessary to calculate the total nitrogen in the feces as well as in the urine, and for two reasons: first, in ordinary diets a certain proportion of vegetable and animal proteid escapes digestion, and this amount must be estimated and deducted from the total nitrogen intake in order to ascertain what nitrogenous material has actually been used up; second, the secretions of the alimentary canal contain a certain amount of nitrogenous material which represents a genuine excretion and should be included in estimations of the total proteid destruction. Practical experience has shown that in man about 29 per cent. of the total nitrogen of the feces has this latter origin.

During the first period the boy was fed on a fluid diet of milk, coffee, cocoa, and water, but during the second he received a much more nitrogenous diet of soup, milk, egg, cocoa, and rice.

It will be seen on close examination that the table shows us marked increase of nitrogen excretion in the later period over the former,

[1] Contributo allo studio del ricambio materiale nel tetano. Revista critica di clinica medica, 1903, Nos. 48, 49, 50.

				Solids.		Sulphates.						
Date.	Amount.	Sp. gravity.	Acidity.	Total.	Inorganic.	Preformed.	Ethereal.	Total N.	NH_3.	Cl.	P_2O_5.	Hu.
August 13, 1904	560 c.c.	1023	25.959	4.15 %	1.22 %	0.32 %	0.02 %	1.86 %	0.21 %	0.61 %	0.50 %	0.07 %
				23.24	6.832	1.808	0.1202	10.438	1.165	3.443	2.805	0.4116
" 15, "	440 "	1027	35.0108	6.58 %	1.048 %	0.46 %	0.02 %	2.43 %	0.25 %	0.54 %	0.48 %	0.057 %
				28.97	4.6112	2.034	0.093	10.6814	1.092	2.374	2.112	0.2508
" 16, "	320 "	1029	12.0304	7.37 %	0.95 %	0.57 %	0.26 %	2.56 %	0.23 %	0.15 %	0.36 %	0.094 %
				23.58	3.0528	1.8198	0.0856	8.189	0.729	0.145	1.152	0.3008
" 17, "	640 "	1018	18.9216	1.22 %	1.114 %	0.203 %	0.018 %	1.389 %	0.15 %	0.74 %	0.18 %	0.05 %
				27.03	7.1296	1.297	0.1172	8.8883	0.097	1.77	1.152	0.317
" 22, "	600 "	1019	alkaline	2.95 %	1.71 %	0.20 %	0.013 %	0.52 %	0.47 %	0.40 %	0.34 %	0.001 %
				17.70	10.26	1.201	0.076	3.108	2.815	2.385	2.01	0.0225
" 23, "	680 "	1021	14.892	4.15 %	1.78 %	0.25 %	0.015 %	1.09 %	0.08 %	0.50 %	0.27 %	0.06 %
				28.22	12.101	1.699	0.099	7.4256	0.566	3.428	1.836	0.408
" 24, "	1060 "	1014	11.607	3.06 %	1.49 %	0.11 %	0.01 %	0.85 %	0.046 %	0.34 %	0.185 %	0.025 %
				32.92	15.815	1.52	0.1323	8.9631	0.486	3.612	1.961	0.262
" 25, "	1040 "	1011	15.184	2.37 %	0.986 %	0.15 %	0.01 %	0.01 %	0.056 %	0.30 %	0.20 %	0.007 %
				24.606	10.254	1.574	0.1106	11.066	0.587	3.14	2.10	0.0707

despite the fact of an increase of nitrogenous foodstuffs, signifying that the boy was already convalescing and a probable restoration of the "nitrogen equilibrium."

The solids were diminished throughout; sulphates but slightly diminished except on the second day, when the patient received some "salts" for catharsis; ammonia varied, at times increased or normal; chlorine diminished; phosphates but little changed; uric acid much diminished.

The urine was at first scant, concentrated, and hyperacid; later, more copious and less acid. A faint trace of albumin was present only at the first examination, never any casts. Sugar was absent.

The figures from this one case compare favorably in a general way with those observed by Vannini in his five cases.

As yet too few practical clinical deductions can be obtained from the study of the metabolism in tetanus, because of the very little that has heretofore been accomplished in this direction, and compells us to trust to the future, with an increase of material and improvement of scientific investigations.

EPITHELIOMA OF THE VULVA.

A REPORT OF SIX CASES, WITH A REVIEW OF THE LITERATURE.*

By Howard Dittrick, M.B.,

LATE RESIDENT GYNECOLOGIST, LAKESIDE HOSPITAL; DEMONSTRATOR IN GYNECOLOGY, WESTERN RESERVE UNIVERSITY, AND ASSISTANT IN GYNECOLOGY, LAKESIDE HOSPITAL DISPENSARY, CLEVELAND, OHIO.

There are many essays on this subject, but the majority consist in a citation of cases with a very brief reference to the literature. Among the German and French writers, such men as Franke, Schwarz, and Maurel have produced excellent monographs, and from these works a considerable amount of the material for this paper has been derived. An attempt has been made to cover everything of importance in the entire literature, with a more minute analysis of the following 135 cases: Mayer,[37] 4; Arnott,[12] 2; Storer,[53] 1; Cushier,[11] 1; Aschenborn,[3] 2; Küstner,[30] 5; Simmons,[52] 2; Rupprecht,[49] 8; Manley,[34] 2; Janvrin,[27] 1; Maurel,[36] 27; Munde,[40] 1; Schmidt,[50] 2; Zeiss,[59] 1; Schwarz,[51] 23; Ingerman-Amitin,[26] 7; Leech,[32] 1; Hirst,[25] 2; Hart,[19] 3; Henry,[20] 1; Mracek,[39] 1; Kelly,[29] 4; Franke,[14] 16; Noble,[41] [42] 3; Butlin,[8] 3; Congdon,[10] 1; Peterson,[47] 4; Reed,[48] 1; Dittrick, 6.

The histories of the 6 cases that have been met with in the Lakeside Hospital will be given somewhat in detail:

* From the Gynecological and Pathological Departments of the Lakeside Hospital.

CASE I.—M. A. B., admitted to Lakeside Hospital August 17, 1898. The patient is sixty-eight years of age and has been married forty-one years. She has borne six children. One sister has "ulceration of the womb," but there is no other history bearing on malignancy in her family. Several months before admission the left labium majus began to enlarge; a nodule, which when first seen was the size of a bean and very hard, has been gradually growing larger. About three months later ulceration began. The discharge from it has been purulent, watery, and bloody. The growth has been more rapid during the last few months. The patient lost twenty pounds in weight during the summer. She has no discomfort with urination, and the urinary examination is negative. The treatment has consisted of local applications of salves.

On examination there is a bloody discharge issuing from the vulval tumor. On the left labium majus is a raw, ragged enlargement pushing the lips apart. The left inguinal glands are indurated and enlarged to the size of the end of the thumb.

The vulval tumor, with a considerable amount of surrounding tissue and the left inguinal glands, were removed by Dr. Dudley P. Allen.

Microscopic Examination. A considerable amount of purulent exudate is seen on the surface. The squamous epithelium is practically normal at one end of the section, but gradually becomes thicker, the cells dipping down into the underlying connective tissue in solid, branching processes. In the centre of the tumor there is a network of larger and smaller masses of these cells. Between the alveoli very little connective tissue is seen, and this is markedly infiltrated with leukocytes. The epithelial cells vary in size, staining reaction, and in the number of nuclei present. Nuclear figures are not numerous. There are many epithelial pearls seen. Many of the epithelial processes are necrotic.

A section through the mass removed from the groin shows a similar picture, with many epithelial pearls.

Diagnosis. Epithelioma vulvæ (cancroid), with metastases in the left inguinal glands. The patient had a recurrence in both inguinal regions six months later, and died as a result of hemorrhage from the left femoral vein August 1, 1899.

CASE II.—R. K., aged sixty-two years, widow. Admitted February 21, 1899. The family history is negative. She has had five children; the youngest being thirty years old. Menstruation ceased at forty-five years, and since then there has been no discharge. The urinary examination is negative.

Four months ago the patient noticed a pimple on the anterior surface of the right labium majus, which gradually increased in size There was constant irritation about the nodule, and she frequently scratched it to relieve the itching. Two months later the surface became ulcerated. During the last month a small ulcer has appeared

on the left labium at the point of contact with the larger ulcer. For three months micturition has been accompanied by intense pain.

On examination on the anterior surface of the right labium majus is an ulcerated surface, 1.5 cm. in diameter, elevated, extending inward toward the clitoris, with an area of induration about 4 cm. in diameter. This is very sensitive. Opposite this on the left labium majus is a small indurated ulcer. There is no apparent involvement of the urinary meatus.

The patient was operated upon by Dr. Allen. An elliptical incision, 10 cm. in length, was made, including both ulcerated areas, extending well up over the mons veneris and down through the labia minora almost to the level of the urethral orifice. The wound healed primarily on the right and by granulation on the left side.

The patient was discharged April 8, 1899, and readmitted July 14, 1899. She had noticed five weeks previously a lump in the right groin, which had steadily increased in size. This was very sensitive, and about the size of the little finger. There was also a small recurrence on the vulva. The glands in the right groin were resected together with the local recurrence. The vulval wound healed primarily. The inguinal wound healed by granulation and was discharging slightly when the patient left the hospital.

Microscopic Examination. The surface is covered with necrotic debris over an irregular base of epithelial cells, which send irregular branching processes into the underlying connective tissue. These cells vary greatly in size, some being ten times as large as others; in some the nuclei are round, in others oval. Some nuclei are small and stain deeply, while others are large, pale, and vesicular. Some cells have one nucleus; others have as many as four nuclei. Nuclear figures are not abundant, but several atypical forms are seen. Many cells show a keratohyaline degeneration. There are not many epithelial pearls. Some of the alveolar processes of these cells are very thick, while others are not more than two cells in width. Many large irregular masses of chromatin are seen in the cell nests. The connective tissue between the epithelial masses is scanty, embryonic in character, and densely infiltrated with leukocytes. Many areas of necrosis are seen in the connective tissues and also in the epithelial cell nests. The bloodvessels are numerous and have very thin walls.

Sections taken from the vulva and from the glands removed at the second operation show the same condition.

Diagnosis. Epithelioma vulvæ, with metastases in the right inguinal glands.

CASE III.—A. W., married, aged seventy-three years. Admitted September 16, 1903. The family history is negative. She first noticed a tumor on the right labium majus seven months before coming to the hospital. Under treatment this would heal over and

then break down. It has been growing progressively larger, and from it there exudes an irritating seropurulent discharge. She has noticed a lump in the right groin for the last three weeks. On examination there is seen to be an irregular, hard, ulcerated, elevated, reddish patch of tissue, measuring 4 cm. by 2 cm., on the right labium majus. On the left labium there is a reddened area which has not ulcerated. On September 18 Dr. Allen excised the tumor and the surrounding tissue, together with the right inguinal glands. One of the latter was necrotic in the centre. The vulval wound healed readily, and the incision in the groin, which was drained, rapidly granulated in and was almost closed when the patient was discharged, September 28, 1903.

Microscopic Examination. The tumor is made up of masses of epithelial cells, invading the entire connective tissue. The cells have the irregular appearance belonging to an epithelioma. Epithelial pearls are very numerous, as many as fifteen being seen in one field under the low power. Many areas of necrosis are seen in the alveolar processes and also in the connective tissue. Many cell nests have dropped out of the section.

Sections through the inguinal glands show considerable necrosis. The glands are extensively infiltrated with the epitheliomatous growth, which shows the same marked pearl formation.

Diagnosis. Epithelioma vulvæ (cancroid), with metastases in the inguinal glands.

CASE IV.—B. K., single, aged fifty years. Admitted December 31, 1903. Menstruation was normal and ceased at forty-six years. In August, 1902, she noticed a growth on the right labium majus. Two months later this was removed, together with the right inguinal glands. Later the right groin became tender and painful, and in August, 1903, the swelling broke, and there has been a discharge from this region from that time. In September, 1903, she noticed a recurrence on the vulva (Fig. 1). She complains also of dysuria, insomnia, and pain in the thigh.

Examination. The right thigh is flexed and cannot be completely extended, while the entire limb is œdematous. Extending along Poupart's ligament, just beneath the skin, is an irregular nodular mass. In the centre of this mass is a small papilla, from which a fetid purulent discharge exudes. The greater portion of the right labium is missing. On the lower portion of the clitoris, just above the old scar, there is a small, plaque-like tumor mass, 2.5 cm. in diameter, and elevated 6 cm. above the surface. This is indurated, ulcerated, reddish, and granular in appearance, with small, whitish areas of necrosis over the surface. There is also a small, granular, ulcerated area in the left labium, where the latter comes in contact with the tumor.

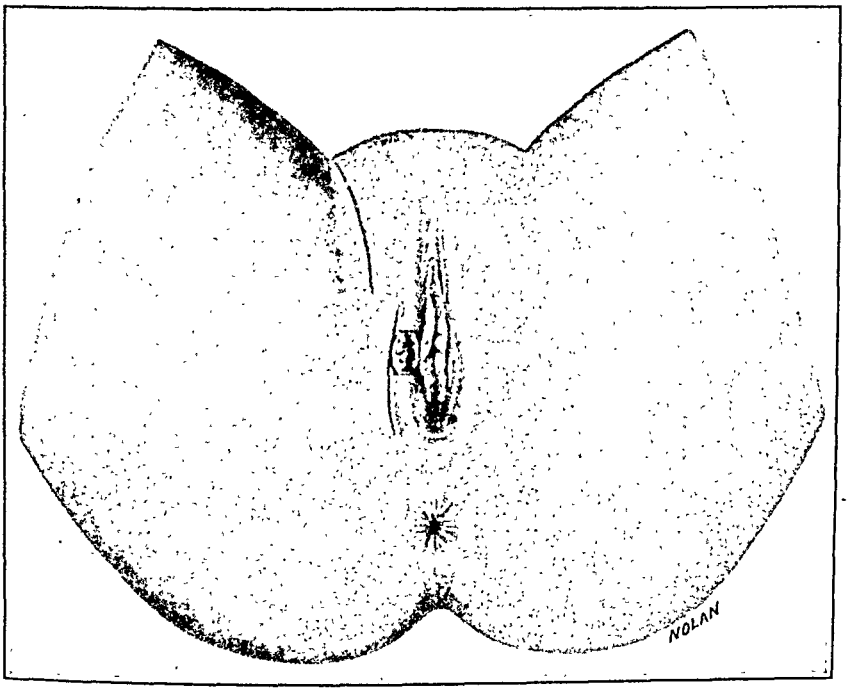
A small portion of the tumor was removed for examination, and an incision made into the mass in the groin to provide better drainage.

In the latter situation a large cavity was found, which extended very close to the femoral vessels.

While the patient was in the hospital several nodules developed in the skin. Two of these were in the abdominal wall, 3 cm. above the mass in the groin, while five were seen scattered over the front and inner side of the right thigh, from 3 cm. to 15 cm. below Poupart's ligament. These nodules were hard, irregular, and reddened, from 0.5 cm. to 1 cm. in diameter, and did not move beneath the skin.

The groin was dressed daily, and three times a week, for two months, the patient was submitted to the x-rays. This procedure

FIG. 1



Scar from previous operation on vulva with small recurrence beside it. Old scar in right groin, showing the papilla from which pus was discharging.

had very little effect on the discharge, and the pain still continued as before. The labial tumor, however, gradually became less prominent, and was almost level with the adjoining skin when the patient was discharged. The process beneath the skin did not seem to be at all checked. The patient left, against advice, on March 5, 1904, and died April 16, 1904, from exhaustion.

Microscopic Examination. The tumor is made up of masses of large irregular cells, varying in size and staining properties. The nuclei are large and oval, with large granules and one or more nucleoli. Typical and atypical karyokinetic figures are seen, but these are not numerous. A few prickle cells are seen in the cell nests. The epithelial cells are found in small collections in the

connective tissue beyond the margins of the large processes. Epithelial pearls are very numerous. The connective tissue is very scanty, but shows a marked infiltration, with eosinophiles, polymorphonuclear leukocytes, and plasma cells. A few small areas of necrosis are seen.

Unfortunately, no sections were obtained from the lymphatic glands in the groin and the skin nodules, but these were undoubtedly metastatic deposits.

Diagnosis. Epithelioma vulvæ (cancroid), with metastases in the right inguinal glands and in the skin over the thigh.

CASE V.—E. G., married, aged thirty-two years. Admitted October 7, 1903. She complained of dull pains in both iliac regions, bearing-down pains in the pelvis, and a tumor on the vulva. The pelvic symptoms were due to adherent tubes and ovaries, with retroversion of the uterus. The family and personal histories were negative. About one year before admission she had noticed a "pimple" on the right side of the vulva. This has gradually grown larger and become painful. When irritated it would bleed. Caustics only stimulated its growth.

Examination. The inguinal glands of the outer group are just palpable on either side. On the inner surface of the right labium majus, rather low down, is a circular, fungating growth elevated 0.75 cm. above the surrounding level. It is granular and eroded on the surface. It is hard and apparently does not infiltrate the surrounding tissues (Fig. 2).

The patient was operated upon by Dr. W. H. Weir and the tumor removed by a wide excision. At the same time the adhesions about the tubes and ovaries were separated and the uterus was suspended after the Kelly method. She made an uninterrupted recovery, both wounds healing by primary union. She was discharged November 7, 1903.

Re-admitted May 5, 1904. One month before this a slight swelling had been detected in the right groin, which had gradually increased in size.

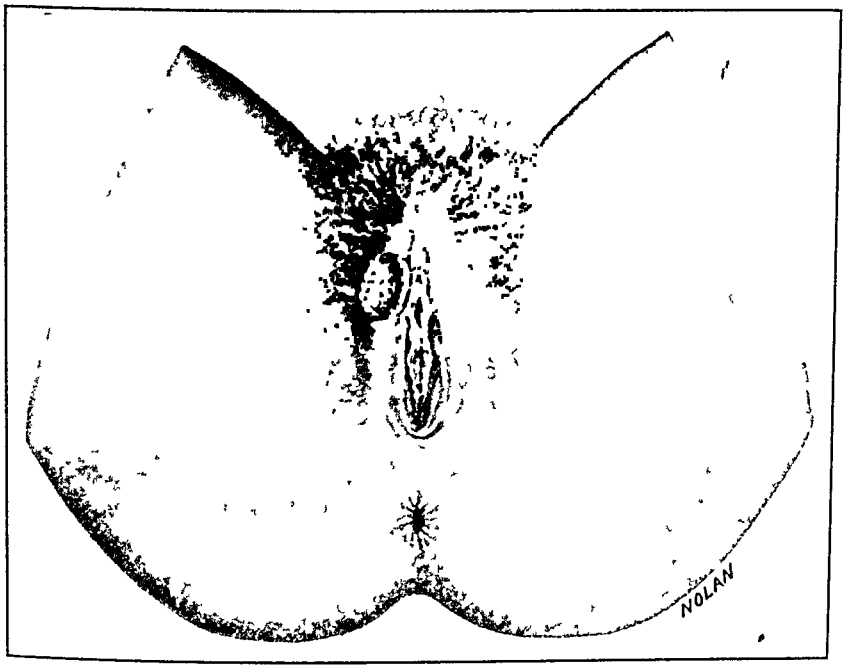
Examination. The outer group of inguinal glands are small and just palpable, while of the internal group one is markedly enlarged, measuring 2 cm. by 1.6 cm. There is no recurrence on the vulva.

The right labium majus and minus were excised. An extensive dissection of the glands and surrounding fat was carried out. A large gland, the size of a hazel-nut, was removed. Another, about the size of a pea, was found along the inner border of the femoral vein. In removing this the femoral vein was wounded, requiring the suturing of the wall at one point. All the fatty tissue was removed as far out as the inner border of the sartorius muscle and as deep down as the femoral vessels. The wound, which was closed without drainage, healed readily, except at the upper angle, where union took place by granulation.

Convalescence was slow, on account of pain down the leg, but this had almost disappeared when she was discharged June 22, 1904. When the patient was last seen (September, 1904) there was no sign of recurrence.

Microscopically. The gradual change from the normal skin to epithelioma is seen. The tumor has not infiltrated as far as the cut margin of the section. It is made up of finely branching cylindrical processes of epithelial cells. The cells have the irregular characteristics of malignancy. Numerous nuclear figures, both typical and atypical, are seen. Many areas of necrosis are apparent. The connective tissue is scanty, except at the margins of the sections. It is markedly infiltrated with leukocytes, many of which are eosinophiles. Epithelial pearls are numerous.

FIG. 2



Epitheliomatous nodule on the right labium majus

The sections removed at the second operation show the same process in the large inguinal gland, while all other sections are free.

Diagnosis. Epithelioma vulvæ, with metastases in the right inguinal glands.

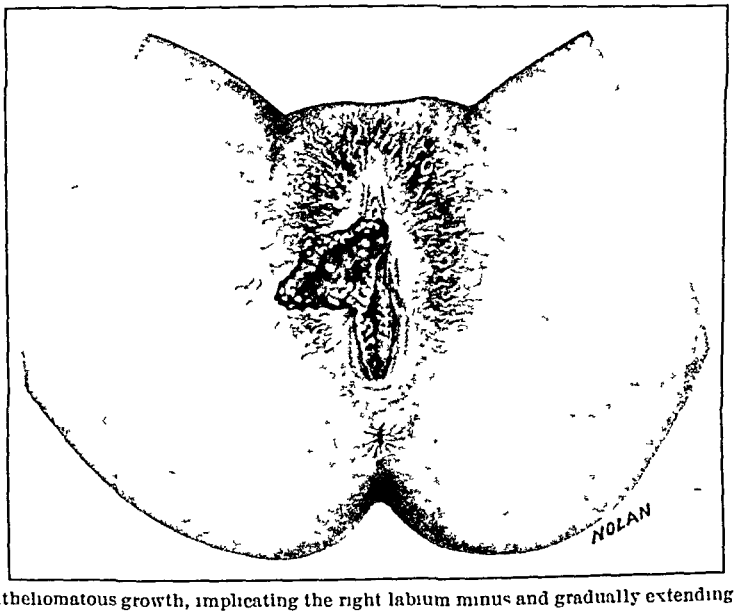
CASE VI.—L. J., widow, aged eighty-one years. Admitted June 22, 1903, complaining of an offensive vaginal discharge, intense itching about the vulva, and a sensitive tumor in this region. The discharge is at times bloody and urination is frequent and smarting. The family and previous histories are negative. There has been a small nodule on the vulva for twenty years. The only treatment has consisted in cleansing lotions.

The patient is exceedingly well preserved. The inguinal glands

are not palpable. The right labium minus presents an irregular, ulcerated, thickened area of tissue, measuring 2 cm. by 1.5 cm. This is sharply defined from the skin. The mass extends from the lower, inner edge of the labium minus to the urethral orifice. The clitoris is not involved. On manipulation the tissue bleeds readily, and small, whitish masses can be squeezed out on pressure. There is no involvement of the vagina or uterus (Fig. 3).

On June 25th Dr. Hunter Robb excised the tumor mass, together with a considerable portion of the right labium majus. The patient stood the operation well. The upper margins of the wound separated slightly, but healed by granulation. When the patient was discharged, July 25th, the wound was slightly congested and indurated.

FIG. 3



Epithelomatous growth, implicating the right labium minus and gradually extending

Re-admitted November 2, 1903. Recurrence has taken place at the site of the old operation. She complains of sharp, darting pains *radiating down the thigh; hemorrhage, profuse on one occasion;* insomnia and dysuria. At the site of the right labium minus is an elevated ulcerated mass, 7.5 cm. by 2 cm. The left labium minus shows two distinct areas of ulceration.

On November 5th the mass was curetted and cauterized by Dr. Robb. This decreased the amount of discharge, and the pain was less severe. Two weeks later the growth again became active, the discharge being more profuse.

On December 22d the mass measured 9 cm. by 3 cm. Under anæsthesia the tumor was excised with considerable surrounding

tissue. This was done in order to have as much of the disease as possible removed before beginning the *x*-ray treatment.

January 4, 1904. There is a large excavated area on the right side of the vulva, measuring 7 cm. by 5 cm. by 5 cm. The outer wall of this cavity is made up of muscular tissue, normal in appearance, while from the floor and inner wall four irregular, fungoid masses are projecting. There is also a small metastatic nodule on the posterior commissure. At this time the *x*-ray treatment was begun. For three weeks this afforded marked relief. Immediately after the treatment the patient enjoyed from one to two hours' sleep, and was free from pain for from six to twelve hours. The large fungoid processes at first became blanched and then gradually subsided to the level of the wall of the cavity. The discharge became less profuse.

April 6th. *x*-ray treatment has been continued every other day. The skin about the growth is much thickened and browned from the treatments. The outline of the excavated area is irregular, and projections from the floor and walls are seen. The left labium is much enlarged and forms a hard, indurated mass, measuring 6 cm. by 2 cm. From the upper angle of this mass springs a hard, irregular growth, which extends over the abdominal wall above the symphysis.

9th. The patient was anæsthetized and a wide excision of the growth was made. The whole mass on the right side, which was adherent to the periosteum, was dissected off the bone. The left labium majus and labium minus and a considerable portion of the mons veneris were removed. A portion of the anterior vaginal wall was also removed from the right side. The wound was partially closed, drainage being employed above and below. Considerable inflammatory reaction followed, which was greatly relieved by daily hot sitz baths.

June 24th. The *x*-ray treatment has been continued, and seems to check the superficial growth, but beneath the skin the infiltration has progressed unabated. The *x*-ray does not now relieve the pain.

There is now an ulcerated area, measuring 11 cm. by 2.5 cm., at its widest point, over the right side of the vulva. There is a small sinus at the upper angle leading to a hard, infiltrated mass above the symphysis pubis.

The nodule on the posterior commissure is slightly larger; the ulceration in the vagina is still limited to the outer third of the anterior vaginal wall on the right side. The inguinal glands are not palpable. The patient was discharged June 24, 1904.

Re-admitted August 12, 1904. She is more cachectic. There is a rounded, sensitive mass over the symphysis pubis. The ulcerated area on the vulva extends 4 cm. into the vagina.

September 13th. The rounded mass over the symphysis (which had reached the size of a cricket-ball) was excised. It was found to be necrotic in the centre.

October 1st. The patient is daily becoming weaker. Her appetite is still good. Her pain is being controlled by morphine. The rectum, urethra, and inguinal glands are still free, and no evidence of metastases can be found in any other organs.

Patient died February 11, 1905. The autopsy revealed an extension along the deep inguinal and iliac glands on either side as high up as the junction of the common iliac arteries. Metastatic nodules were found in the bladder, the stomach, both kidneys, both lungs, left pleura, and the heart. This was confirmed by microscopic examination.

Microscopically. The tumor is made up of finely branching processes of epithelial cells, which are irregular in size and staining properties. There are from one to four nuclei in the cells, vesicular or deeply stained, and containing from one to four nucleoli. There are no epithelial pearls. Both regular and irregular mitotic figures are seen, although they are not numerous. The connective tissue is infiltrated for a considerable distance beyond the margin of the tumor. Many of the connective-tissue cells have deeply staining nuclei, but none show nuclear division. The connective tissue is extensively infiltrated with leukocytes. Tissue from the second operation shows irregular epithelial processes made up of large cells, which in most places have oval or round, vesicular nuclei. There are numerous concentric cell nests with pale centres, but these are not as hyaline in appearance as in epithelial pearls. Tissue from the third operation shows a few epithelial pearls, while nuclear figures are more abundant. There is very little stroma between the epithelial processes. Tissue from the later operations shows the same features as in the last specimen.

Diagnosis. Epithelioma vulvæ.

FREQUENCY. Cancer of the external genitalia of women is a rare disease. Franke, in May, 1898, found records of 15 cases primary in the clitoris. Veit[55] refers to 4 cases primary in Bartholin's gland. Percy[46] has recently collected 16 cases of primary carcinoma of the urethra. Sarcoma, melanosarcoma, myxosarcoma, fibrosarcoma occurring on the vulva are relatively rare.

According to Virchow,[56] cancer occurs in men and women in the proportion of 9 to 11. Williqks,[58] in 5536 autopsies, found cancer in 477; 190 of the subjects being males and 287 females. Gusserow[14] met with carcinoma in men and women in the proportion of 25 to 61. Among 8500 female patients, L. Mayer[38] had 332 malignant tumors, distributed as follows: 119, or 1.5 per cent., of the uterus, predominating in the cervix; 146, or 1.7 per cent., of the uterus, with involvement of the vagina; 256, or 3.2 per cent., of the uterus; 8, or 0.09 per cent., involving the vagina without the uterus; 10, or 0.1 per cent., of the vulva; 2, or 0.02 per cent., of the ovary; 8, or 0.08 per cent., of the breast; 39, or 0.45 per cent., in other than the sexual organs.

Gürlt[18] arranged the following table indicating the incidence in 11,140 benign and malignant tumors in females, 7479 of which were carcinomatous. Of these, 2845 were in the uterus alone; 604 in the uterus and vagina; 114 in the vagina alone; 81 in the vulva; 71 in the ovary.

Schwarz met with 1 vulval carcinoma to every 38 uterine carcinoma, Virchow[56] 1 to 35 to 40, and Gürlt[51] 1 to every 48 in the uterus.

Gönner[17] found, among 900 gynecological patients from 1873 to 1881, 99 with carcinoma, and in 9 of these the vulva was the primary seat. Eisenhart[13] found, among 658 gynecological patients, 2 with primary vulval carcinoma.

Billroth,[6] in eight years, met with 4 vulval carcinomata among 548 cancer cases.

In 2134 gynecological patients admitted to Lakeside Hospital, there have been 106 cases of carcinoma of the generative organs, distributed as follows:

Cervix uteri	72
Fundus uteri	8
Vagina	2
Ovary	18
Vulva	6
	106

Carcinoma of the vulva has occurred in 0.28 per cent. of our gynecological patients, and forms 5.66 per cent. of the cases of carcinoma of the female generative organs. These figures represent fairly accurately the relative frequency of this condition, for by combining the cases reported by Mayer, Gönner, Eisenhart, and those presented in this paper, 27 cases, or 0.22 per cent., were found in 12,192 gynecological patients.

According to Gürlt,[18] carcinoma of the vulva formed 10 per cent. of all cancers in the female. His findings are based on observations covering 11,140 benign and malignant tumors.

AGE. Sarcoma of the vulva occurs at an earlier age than carcinoma. Launois,[36] however, reported a case of sarcoma in a woman aged sixty-two years.

Carcinoma, on the other hand, occurs at the end of the activity of sexual life. The oldest patient was ninety years, the youngest twenty years, and the average was seventy years. In 84 per cent. the patients were over forty-five years, the usual time for the menopause.

Every author who has written on this subject quotes West's[57] case in a woman, aged thirty-one years, as the earliest appearance of carcinoma of the vulva. There are, however, 4 well-authenticated cases in patients aged thirty,[51] thirty,[41] twenty-nine,[32] and twenty[2] years, respectively, the diagnosis in the second and the fourth having been confirmed by microscopic examination.

In 126 cases in which the age was given they were divided as follows:

Age.	Cases	Per cent
20	1	0.8
21 to 30	3	2 4
31 " 40	12	9 5
41 " 50	24	19 0
51 " 60	28	22 2
61 " 70	41	32 6
71 " 80	14	11.1
81 " 90	3	2 4
	126	100 0

Winckel[14] gives the following statistics on 54 cases:

Age.	Per cent.
30 to 40	9 7
41 " 50	25 8
51 " 60	32 2
61 " 70	25 8
71 " 75	6.5

Mayer's[14] statistics are:

Age	Per cent.
20 to 30	4 51
31 " 40	22 93
41 " 50	24 19
51 " 60	20 68
61 " 70	9 03
71 " 80	1 13

Mayer finds the greatest frequency in the fifth decade, Winckel in the sixth, and our own figures, based on a much larger number of cases, in the seventh decade. The ages of the individual cases were as follows: Twenty, twenty-nine, thirty (2), thirty-one, thirty-two (2), thirty-four (2), thirty-five, thirty-six, thirty-seven, thirty-eight (2), thirty-nine, forty, forty-three (2), forty-four (2), forty-six (2), forty-seven (5), forty-eight (2), forty-nine (6), fifty (5), fifty-one (3), fifty-two (2), fifty-three, fifty-four (5), fifty-five (4), fifty-eight (6), fifty-nine (2), sixty (5), sixty-one (3), sixty-two (6), sixty-three (2), sixty-four (3), sixty-five (5), sixty-six (4), sixty-seven, sixty-eight (6), sixty-nine (2), seventy (9), seventy-one, seventy two, seventy-three (3), seventy-four (5), seventy-five, seventy-six (2), seventy-eight, eighty-one, eighty-four, ninety.

Etiology. In speaking of the etiology, the cause of cancer in general will not be considered, but only those conditions which predispose to its appearance on the vulva.

The principal factors, as usually quoted, are mechanical injuries and sources of chronic irritation. The latter class includes many and varied conditions; the former has to do with trauma incident to pregnancy, coitus, and accidental injuries. One would think that if direct injury had anything to do with the causation of carcinoma, it would sometimes follow difficult instrumental deliveries; yet in only 3 cases is pregnancy spoken of as a possible causative agent.

Aschenborn[3] details a case that seemed to begin after a labor, while in the second case, cited by Franke, the tumor had been first noticed sixteen years before, at the first confinement, and after each labor it had increased markedly in size. In another of Franke's cases the patient had undergone eleven deliveries with forceps, but the disease was not definitely ascribed to the trauma connected with them. Coitus, too, may be attended by very severe injuries. Nussbaum[44] cites 2 cases in which the abdominal recti muscles were ruptured. Himmelfarb[23] quotes 2 cases of extensive laceration of the vaginal wall, 1 of which was followed by infection and death. Schulein[14] had 2 severe cases of laceration of the hymen. Other cases are mentioned by Bohm,[14] Franke,[14] Zeiss,[14] Teuffel,[14] and Berthel Anderson,[14] but the etiological relation to epithelioma of the vulva is not clear.

West[57] reports a case referred to a fall against the edge of a chair, with profuse hemorrhage five months before. In the monograph by Ingerman-Amitin, a case is described in which the patient fell on a chest and suffered from a considerable tearing of the right labium. Two years later a carcinoma developed at the site of the injury.

Although it can be readily seen that the vulva is exposed very often to severe injuries, very few of these accidents are followed by malignant disease. Since 2 of the cases reviewed in this paper occurred in women in whom the hymen was intact, it is clear that there are other factors that must be taken into account.

Other conditions concern the lowering of the local resistance and the keeping up of a chronic irritation. In looking over the histories of these cases, one of the most striking features is the number in which pruritus has preceded the carcinoma. In 27 cases (20 per cent.) the patients had suffered from this condition for from one month to five years before there was any sign of a tumor.

Of course, in those instances in which the irritation has preceded the tumor by a few months only it must undoubtedly be regarded merely as an early symptom. Where, however, the patient had suffered for several years from an itching, which has given rise to considerable scratching with some abrasion and pigmentation, and later a carcinoma has developed on this area, it is only fair to assume that the pruritus was at least an indirect agent in its production. Where small papillomata, which are frequently met with on the vulva, are associated with a long-standing pruritus, it is not difficult to understand how the constant irritation of scratching could start up a malignant growth. This is well illustrated in Case VI., in which such a tumor had existed for twenty years, and in which, for a long time, there had been an annoying pruritus. For two years this had shown active growth, and at the end of that time was a typical epithelioma.

In a case reported by Noble, pruritus had been present for twenty years, while the patient had only known of the existence of a tumor

a short time. Kelly speaks of a case in which pruritus had been present for four years, and a spot like proud flesh was noted only two months before the patient came to him. Franke thinks that either the senile or diabetic form of pruritus may be followed by carcinoma. This is probably true as regards the senile form. In none of the cases was there a preceding history of a glycosuria, so that diabetic pruritus cannot be considered as a causative factor. But whether pruritus is an etiological factor or merely an early symptom, it should always be remembered that intense and obstinate itching is frequently the forerunner of malignancy, and whenever such a symptom is present a thorough examination with close inspection should be made.

In most essays there is very little mention made of leucoplakia in this connection. Hildebrandt,[51] Schwarz, Butlin, Noto,[43] and Besc[5] have emphasized the fact that leucoplakia of the vulva often precedes carcinoma. The buccal leucoplakia seen in habitual smokers, and variously described as psoriasis, ichthyosis, or keratosis, is not infrequently the antecedent of carcinoma in this region. Similar plaques are seen on the vulva and may be the precursors of epithelioma. These areas are irregular in circumference, opalescent, and pearly in appearance. They are situated on the labia majora or minora, clitoris, or even on the vaginal wall. Very often they are seen about advanced carcinomata and do not tend to disappear. Szasz[54] (Budapest) says that leucoplakia is a natural evolution between kraurosis and carcinoma.

Louis Mayer [51] has demonstrated a case in which carcinoma began in the epithelial thickening in the centre of one of these patches of leucoplakia. Schwarz reports a case in which a cancroid developed on the site where one year previously he had noted a leucoplakial patch about the size of a dollar. The diagnosis was verified by microscopic examination. Jouin[28] cites a case in which white plaques had been present a long time before ulceration began, being found constantly for two years. Franke, Maurel, Schwarz, Kelly, and Butlin have also seen leucoplakia of the vulva and vagina associated with carcinoma of the vulva. Since, then, carcinoma has been known to develop on areas of leucoplakia, they should be watched very carefully, and on the first suggestion of malignancy they should be widely excised.

Psoriasis is also mentioned as a precedent condition. Maurel quotes such a case in the practice of Dr. Saint-Philippe, where for fifteen or twenty years there had been psoriasis of the vulval mucosa, giving rise to some secretion and very painful itching. During the last four months an epithelioma developed, and six months later there were metastases in the inguinal glands.

Brittauer[7] reports a case of kraurosis vulvæ, which, from scratching to relieve the pruritus, became ulcerated, and, according to his description, probably malignant.

Lancial[31] reports a case of primary carcinoma of the left labium majus following a chronic abscess. This reminds one of carcinoma of the breast, which in some instances has been attributed to a chronic mastitis.

Arnott[30] has seen a case associated with syphilitic ulcer. Hutchinson says that old sites of syphilitic areas may become cancerous. Maurel quotes him as follows: "Syphilitic ulceration with inflammation and hypertrophy may degenerate into cancer in a way so imperceptible and gradual that it is impossible to say when one ends and the other begins."

According to Veit,[55] repeated infections with gonorrhœa and syphilis predispose to carcinoma. It is interesting to note, however, that none of the patients ascribed their condition to irritation from a vaginal discharge.

Masturbation associated with pruritus is mentioned by Ingerman-Amitin as preceding carcinoma. Two other cases are mentioned by the same author as being due to continued irritation. In one the condition was ascribed to the chafing from wearing a truss, and in another to continually rubbing the parts against the side of a bed. L. Mayer[37] also had a case in which the origin dates back to irritation from a truss. In this same connection, Schwarz has found similar factors in the production of primary vaginal carcinoma. Zizold[60] reported a case in which the carcinoma developed in a prolapse of the vagina, where the parts had rubbed against the inner surface of the thigh. Hegar,[51] Winckel,[51] and Kaltenbach[51] have reported cases occurring from the pressure of pessaries. Schwarz had a case in which a pessary had remained undisturbed for a year. The woman had a sudden profuse hemorrhage, and on examination the pessary was found completely encrusted by an extensive carcinoma, which had developed in the region of pressure.

Family History. In only one case has any mention been made of malignant disease in the patient's family. The mother of one patient[29] died of cancer of the lip.

Previous History. One patient[36] was treated for six or seven years for a vaginal eruption, which first appeared at twelve years of age. Sixteen years previously a second patient[36] had had eczema of the scalp, followed some months later by icterus. Another patient[47] had had a tumor of the larynx removed eleven years previously, with no return. One patient[36] lived amid inferior hygienic conditions. One patient[14] had had puerperal fever, and two[14] had been curetted.

Associated Conditions. The following associated conditions were found: leucoplakia in 8 cases; retroflexion in 5; retroversion in 5; pelvic peritonitis in 2; psoriasis in 2; descensus uteri, double hernia, cervical polyp, femoral hernia, dropsy, fibroid and ovarian tumors, and adenoma of the right breast in 1 case each.

CHILD-BEARING. There were 97 patients married, 8 single, and 10 widowed. In 59 cases where the number of pregnancies is given, they were divided as follows:

Para	1	2	3	4	5	6	7	8	9	10	11	12
Number of cases	6	9	12	9	4	6	3	4	2	1	1	2

In 1 case there was a history of a twin pregnancy,[30] and in another there was a laceration of the perineum. One patient[14] had had eleven confinements, in all of which the forceps were used.

Occupation does not seem to play any part in this disease. In none of the cases is there any stress laid on this point.

SYMPTOMS. Epithelioma of the vulva may exist for a long time without causing any symptoms, and very often the tumor is only discovered accidentally. In 20 cases the presence of a small tumor was noted as the first evidence of the disease. In the majority of instances (44 in the series) the first sign of the disease was *pruritus*. This is variously described as an itching, burning, pricking, or sticking sensation, coming on in paroxysms, or sometimes constantly present. The discomfort is very intense and gives rise to considerable scratching, producing excoriations and pigmented areas about the vulva. It may disappear entirely for a time and then recur.

Ingerman-Amitin made the following statement: "It is possible that the origin of a small unnoticeable carcinomatous nodule may cause itching, which can exist as the only symptom for a long time, without the patient or even the doctor knowing the true condition."

The pruritus is due to the irritation of the nerve endings of the ilioinguinal and cutaneous branches of the pudic nerves. Noble[42] carefully dissected out these nerves in a case of epithelioma of the clitoris, and the itching was promptly relieved. Schwarz calls attention to the worried and anxious facies due to this uneasiness about the vulva.

Pain is one of the later symptoms, although it was the first cause of complaint in 6 cases. The tumors, when seen early, are usually neither painful nor sensitive, but the pain increases in severity as ulceration advances. The pain is described as sharp, piercing, darting, pricking, lancinating, stinging, smarting, or burning. It comes on in paroxysms, and is worse at night. It frequently radiates into the hip and down the thighs or may be referred to the lower abdomen or the vagina. Walking or even sitting is soon accompanied by great distress, and the patient becomes bedridden. Pain was marked in 46 cases, and in 7 cases there was no pain, but the latter were seen early in the disease. The pain was very severe in 3 cases in which the tumor had been growing for four months, while in others the growth had been present for more than a year.

Urination is nearly always interfered with as the carcinoma advances, owing to the trickling of the urine over the ulcerated surfaces. There was smarting, burning, scalding pain on voiding in

22 instances, and in many of these there was increased frequency. In advanced cases there may be incontinence. There may be difficult micturition from the fact that the surrounding infiltration causes a stenosis. Catheterization may be difficult, and there may be such a degree of retention as to force the patient to submit to operation.

Frequently, from the mere mechanical irritation of the tumor, there is a slight *discharge*, keeping the labia moist and causing discomfort. As soon as ulceration takes place the discharge becomes more profuse. At first it is whitish, mucoid, and watery, but later, as the tissue degenerates, it becomes more fetid, yellowish, and purulent. It is tinged with blood from time to time, and in 3 cases there was profuse hemorrhage. In 1 case *hemorrhage* was the first symptom; 45 patients complained of a discharge. *Painful coitus* was the initial complaint in one patient. An intractable insomnia very often results from the pain and discharge.

COURSE. Maurel describes a pre-tumoral period, which is accompanied by severe and intolerable pruritus, coming on at intervals in women who have passed the climacteric. The condition at the time, he admits, is unrecognizable.

In the second period, a tumor is present with infiltration about its margin, but without any ulceration. This tumor formation begins in two different ways. The first is characterized by a localized area of thickened and whitened tissue, the surface of which becomes cracked and fissured, and from these clefts the epithelial growth may spring. In the second class of cases the patient's attention is first directed to a prominent tumor resembling a "wart," "pimple," "blister," or "proud flesh." One meets with these small, warty prominences on the vulva frequently. Papillomata, Veit says, is the most fitting name for them. They resemble venereal condylomata, their etiology and failure of extension constituting important distinctions. These growths remain quiescent for months or even years, after which, from some cause, they may take a rapid growth and become typical malignant tumors.

Thirdly, we have the period of ulceration, and occasionally only at this time is the tumor first noticed. Involvement of the upper chain of the inguinal glands soon develop. The final stage is much more rapid. The ulceration becomes more marked. Occasionally metastases occur in the viscera. Death from exhaustion is the usual ending.

As a general rule, all the tumors are of slow growth. They may be latent for months or even years, until some accident calls the patient's attention to the vulva, and a tumor is discovered. A tumor had been present on the vulva for twenty, sixteen, and eight years, respectively, in three cases. The patients usually present themselves six or seven months after they have noticed the growth.

As illustrating the tardy growth the following cases are of interest:

In one, after eight years, during the last six months of which the increase had been relatively rapid, the tumor measured 3 cm. by 3 cm. Other cases at the end of a year presented the following appearances: growth, the size of a filbert; ulcer, the size of a bean; three nodules, the size of a bean, 1 cm. in diameter; ten-pfennig-sized tumor; not ulcerated; two small, wart-like nodules.

The following showed a more rapid increase in size. After one year in one patient the growth had encircled the vulval outlet. In eight months another tumor had almost completely obstructed the vulval orifice. Another had increased to the size of a goose's egg in five months. Tumors the size of a hen's egg were found after six months, three months, and three weeks, respectively. In four weeks one tumor had reached the size of a dollar.

The average duration of the disease is about two years without operative interference. In one rapidly growing tumor the patient only lived four months after the tumor was discovered (Deschamps[12]). In other cases the tumor may grow very slowly, and may not cause death for many years. In Case VI. a tumor had been present twenty years, and had grown actively for three and one-half years

DIAGNOSIS. The vulva is the seat of other lesions, which are often difficult to differentiate from epithelioma.

In the early diagnosis, tuberculosis and syphilis are first to be eliminated. In tuberculosis or lupus under treatment the disease tends to heal with cicatrization, and often with considerable hypertrophy of the surrounding tissue. Tuberculosis occurs in younger patients and does not show as great a tendency to bleed on manipulation. When there is any doubt a small fragment should be excised and examined microscopically, as at times this is the only method possible to clear up the diagnosis.

When the disease occurs in the form of a prominent tumor it may resemble myxoma, sarcoma, or syphiloma. Sarcoma occurs early in life and is very rare. Early in the epitheliomatous process there is a close resemblance to the initial lesion of syphilis, but when the surface becomes ulcerated, the characteristic granular surface, the irregular, yellowish-white areas of necrosis, the hard consistency, the indurated border, and the resistance to antisyphilitic treatment, aid in the diagnosis. This feature was studied by Géber[15] in 1871. Four cases in this series were first treated for syphilis.

In the ulcerated form the lesion might be mistaken for a syphilitic or tuberculous ulcer, an ulcerated fibroma, or an ulcerated lipoma Elephantiasis may have papillary excrescences resembling epithelioma.

Peterson reports a case that had been lanced, on the supposition that the growth was an abscess.

Psoriasis, which is indolent at the beginning, with very little itching or sensitiveness, may later be confounded with carcinoma.

When the inguinal glands are palpable, and there is rapid growth with cachexia, the diagnosis is more easily made.

PROGNOSIS. The prognosis is very grave. The fact that it occurs in women of advanced age, frequently limits the treatment to palliative measures, on account of other co-existing conditions, which contraindicate a radical operation. When the disease is seen before the inguinal glands are involved, the prognosis, of course, is more favorable. A radical operation at this time may give the patient several years of absolute freedom from the disease, although at any time it is likely to return.

PATHOLOGICAL ANATOMY. The primary seat may be on any of the structures comprising the vulva. In this series the following were first involved:

Labium majus	52 cases.
Clitoris	35 "
Labium minus	21 "
Anterior commissure	3 "
Posterior commissure	3 "
Vestibule	1 "

In 53 cases the disease began on the right and in 31 on the left side. The disease most frequently attacks the labium majus on the inner surface of its posterior half or in the sulcus between it and the labium minus.

In the beginning the disease occurs as a small warty tumor, a localized thickening, or even as a small ulcer. Later when the disease is more marked, Gebhard[16] recognizes three distinct types: (*a*) a prominent tumor; (*b*) a diffuse infiltration which is not elevated; (*c*) deep, crater-like excavations. Maurel describes two typical forms: (*a*) vegetating; (*b*) infiltrating. These two forms may co-exist.

In looking over 123 described cases, two-thirds were found to have occurred as a prominent tumor formation. In 23 per cent. there was a carcinomatous ulcer; 7 per cent. showed a crater-like excavation; 4 per cent. appeared as the diffuse carcinomatous infiltration spoken of by Gebhard. Two cases in the last class, when seen some months later, had developed eaten-out ulcers. The carcinomatous ulcers, through infiltration into the deeper tissues, with accompanying necrosis, later developed into necrotic cavities. Consequently when the disease is well advanced there are but two classes: those in which there is a prominent vegetating tumor and those in which there is ulceration, infiltration, and necrosis, extending deep into the cellular tissue.

In the different cases the disease presented the following appearances:

Duration of growth.	Prominent tumor formation.	Diffuse infiltration.	Depressed ulcer.	Crater-like excavation.
Six months or less	29	3	12	3
Six months to one year	17	...	6	2
Over one year	9	...	2	2
Time not stated	25	2	9	2
Total	80	5	29	9

About the site of the lesion the skin is frequently excoriated and pigmented from scratching, and plaques of leucoplakia are often seen. The larger number of cases are first seen when the process is represented by a prominent tumor. This varies with the progress of the disease from the size of a small bean, until it may completely block the vulval orifice. At first it is smooth, rounded, yellowish-white, sharply circumscribed, and freely movable over the underlying tissues. Occasionally it forms a flat, plaque-like tumor. Ulceration may be present as early as three weeks after the tumor has been noticed, or, in a small number of cases, there may be no ulceration for years. Usually, however, it is present at the end of six months. The ulceration begins on the inner and lower surface of the tumor, where it comes in contact with the structures of the opposite side. After this change has taken place growth is more rapid. The surface becomes irregular, uneven, raw, granular in appearance, and the color varies from pink to dark red or even violet. It may be nodular or fissured, and in the crypts between the small prominences are whitish collections of pus and epithelial debris. Small localized areas of necrosis give it the appearance of an erosion produced by an acid. The growth may be papillary or fungoid and consist of numerous densely packed papillæ, with mushroom-like edges. In consistence it is very hard and firm, except where there is much necrosis, when softish areas are felt. A more or less thickened pedicle may be present, especially when the growth arises from the clitoris.

The tumor bleeds readily on manipulation and is very friable. On section it is pearly white, with small yellowish areas of necrosis, which can be squeezed out on pressure. At first the tumor is superficial; later epithelial cords are felt extending into the deeper tissues through the lymph channels; the base is hard and indurated. Finally the growth becomes adherent to the os pubis and is firmly fixed.

More rarely the growth begins as a diffuse infiltration with no elevation above the surrounding tissue. It soon becomes ulcerated, and presents the usual appearance of a carcinomatous ulcer. When first seen the ulcer is usually about the size of a twenty-five-cent piece, and the growth is freely movable. The tissue about and beneath it becomes indurated and infiltrated, and the ulceration in the end involves one or both sides of the vulval orifice. The edges of the ulceration are irregular, elevated, and undermined. The base is reddened, moist, fissured, and has an eaten-out appearance. From the base papillary fungoid masses arise. Areas of necrosis are seen as whitish spots irregularly distributed over the floor.

The crater-like form is merely a more extensive degree of ulceration, in which the centre is deeply excavated from the penetration of the carcinoma into the underlying connective tissue.

Microscopically. Epitheliomatous growths of the vulva are divided into four classes: scirrhus and medullary carcinoma, cancroid and

melanocarcinoma. The first two varieties are distinguished solely by the amount of intervening connective tissue; in the former the stroma; in the latter the epithelial tissue preponderates. L. Mayer[51] published 2 cases of the scirrhus variety. The microscopic descriptions are so few in the literature that it is impossible to tell how many cases are medullary in type. Four cases, 1 reported by Percheron[45] and 3 by Franke, seem to belong to this class. Cancroid is the term which the German authors use to represent the variety characterized by the presence of numerous epithelial pearls. Schwarz reported 23 cases. (This class forms the greatest number of epitheliomata of the vulva.) Deschamps[12] reported an instance of degenerating epithelioma in which some of the areas resembled colloid material. Numerous pearls were also seen, which would lead one to believe that it was a degenerating cancroid. Melanocarcinoma is characterized by the presence of brown or black pigment particles, either in the cancer cells or in the tissue about them. Bailly[1] and Franke have each reported a case of this kind. None of the cases belong to the glandular form of carcinoma, unless we include those in which the growth originates in Bartholin's gland. In 73 cases of this series the diagnosis was verified by microscopic examination, but in the others the descriptions, plates, and clinical histories show that they were undoubtedly epitheliomata. The progress takes place by direct extension along the lymph spaces, with degeneration of the adjoining tissue. In this manner the cutaneous tissues about the vulval orifice may be involved from the mons veneris back to the rectum.

Metastatic nodules are not infrequently found in the neighboring areas of the vulva or even back on the perineum, the infective material having been carried through the lymphatics, although in the latter case the cells have travelled against the lymph current. An interesting feature is also met with in cases in which the surface of the opposite labium, in contact with the tumor, becomes the seat of carcinoma. Cases of this nature are cited by Schwarz, Hildebrandt,[22] Zweifel,[56] and Kelly. Franke points out the origin as due to the invasion of the cancer cells through the blood or lymphatic vessels; mechanical irritation of the tumor itself; lowering of the local resistance from the secretions of the ulcerated tumor; direct transplantation of masses of cells; and the implantation of the organisms, if such there be, of cancer. There is no doubt that similar tumors arise at the point of contact, but the etiology in these cases is a matter of theory, and probably all but one of the causes assigned by Franke play a part. The parasitic theory, however, with our present knowledge is untenable.

Hildebrandt has said that carcinoma of the vulva only exceptionally extends beyond the caruncule myrtiformes and the margin of the vulva. Schwarz says that conversely primary vaginal carcinoma rarely encroaches upon the vulva. In 14 cases the vaginal wall was

involved, and in 2 cases[36] [51] the disease had extended along the entire length of the posterior wall and finally invaded the cervix uteri.

The urethral orifice is rarely invaded, but in 1 patient[50] the carcinoma had involved the urethra and the neck of the bladder. The cutaneous tissue, as far as the groove of the groin, had become infiltrated in 2 cases. The infiltration in 6 cases had extended upward beneath the mons veneris, and in 2 cases over the anterior abdominal wall. The clitoris was secondarily invaded in 17 cases, and the opposite side of the vulva in 11 cases. Small metastatic nodules are found in 2 cases in the skin over the inner surface of the thigh.[9] [36]

Metastases in the inguinal glands are very common. Through the communication of these with the iliac, sacral, and lumbar glands, metastases may be found in any of these groups. Percheron[45] describes such a case in which the inguinal glands were enlarged, and similar nodules were found along the iliac vessels and on up to the right of the aorta. In this case numerous nodules were also found in the lower part of the abdomen and on the posterior surface of the uterus. Cases in which the disease had involved the abdominal lymph glands are also reported by Küstner, Franke, Hirigoyer,[24] Saint-Philippe,[36] Schwarz, and Ingerman-Amitin.

Cases are reported in which other viscera have shown metastases, infection having probably occurred through the blood stream. In a case reported by Zeiss[59] there was a carcinomatous nodule in the left breast, with two infiltrated axillary glands. Küstner's[30] patient had miliary carcinosis of the lung, with carcinoma of the heart, liver, spleen, and kidney. Cancerous nodules were found by Arnott in the pleuræ, in both lungs and in the heart, the primary seat being on the clitoris. Franke had a case in which, from the clinical symptoms, he diagnosed metastases in the lungs. Léger[33] found in his patient cancerous nodules in the kidneys and lungs.

In those cases in which the glands were found to be carcinomatous, 50 per cent. had become involved during the second six months of the tumor's existence. One case showed involvement of the glands at the end of three months, another three and one-half months, and another in four months. On the other hand, a case reported by Frerichs[14] showed well-marked ulceration for three years with no glandular involvement.

In 33 cases clinically there was involvement of the glands, and in 9 of these the diagnosis was confirmed by microscopic examination. In 36 additional cases the glands were enlarged, but in 12 of these the condition was due to hyperplasia of the lymphoid tissue, without any carcinomatous invasion. Several cases showed slight enlargement of the glands before excision of the ulcerated tumor, and with the removal of the growth the swelling of the glands subsided. In 28 cases recurrence took place in the inguinal glands after operation. Where the glands from one side only had been removed in 2 cases,

recurrence took place in those of the opposite side. In 7 cases there was a recurrence in the groin after the glands of the same side had been removed.

The disease may be so extensive in the inguinal glands that the blood supply of the leg may be interfered with sufficiently to cause œdema or even gangrene of the limb, as reported by Rupprecht.

TREATMENT. In cases which are seen before the glands have become enlarged a radical operation holds out the greatest hope.

Authorities do not agree on the treatment advised for the inguinal glands. Diffenbach[20] and Hildebrandt[20] hold that the new-growths are inoperable when the lymph glands are swollen. Winckel[20] considers excision of the glands a grave procedure on account of the patient's age, especially when they are bedridden. He asserts that exclusive excision of the primary growth is without doubt satisfactory. He employs this even when the superficial inguinal glands are enlarged, as this swelling usually subsides under such treatment. Other authorities remove the glands only when they become enlarged. Some take out only the glands on the side involved and remove those of the opposite side, if necessary, later. Rupprecht[19] advises removal of the glands on both sides, even though they may not be swollen. Veit says the inguinal glands should be removed before excising the tumor. As recurrence may take place on the opposite side of the vulva, or in the inguinal glands, on either side, it would seem better to take out the glands from both groins as well as both sides of the vulva.

When an excision of the primary growth only is to be carried out, the entire side of the vulva should be removed, but if there is the least suspicion of excoriation or induration, the opposite side should also be excised. The incision should be carried well outside the tumor from the mons veneris above to the posterior portion of the labium majus on either side. On the inner surface the incision is made just outside the carunculæ myrtiformes, the two incisions meeting a short distance above the urethra. Of course, this may have to be altered if the vaginal wall or the urethra is involved, and the incision must be made to include the entire growth. The mass so outlined must be dissected out, and the fatty tissue removed as deep down as the pubic bone. Maurel advises the use of the écraseur and cautery to accomplish this. When completed, Franke and Küstner have an inverted Y-shaped wound.

Rupprecht recommends extensive dissection of all glands, superficial and deep, analogous to the operation for cancer of the breast. His results justify the recommendation of the procedure as the best treatment for epithelioma of the vulva. The incision is carried from the symphysis pubis to the anterior superior spine. A second incision is made downward from the first, along the course of the femoral vessels. The skin is dissected upward to the aponeurosis of the abdominal muscles, outward to the fascia lata, and inward

to the adductor muscles. The entire three-cornered mass of fat and glands is separated down to the deep vessels, after Scarpa's triangle has been cleaned out. The saphenous vein will have to be ligated where it joins the femoral. It is often necessary to divide Poupart's ligament and remove glands from the sheath of the vessels.

When the mass of fat and glands has been thoroughly dissected out on one side the other groin should be treated in a similar manner. These masses may either be removed singly or they may be taken out with the vulval growth in one piece.

In Case V. the femoral vein was wounded and the wall was sutured with silk. Leech tied the femoral vein in one case, and, although marked œdema followed, there was no gangrene. Langenbeck,[49] Bergmann,[49] and Braun[49] have tied the femoral artery. In such cases, Rupprecht advises the maintenance of the leg in the upright position with no compression.

Schwarz cites a case in which, on account of a heart lesion, Olshausen excised the growth and both glands under cocaine.

As a rule, the wounds heal well, but on account of the great tension in some cases there may be some separation of the edges, with healing by granulation. The operation is attended by very little shock, and the patients stand it well. Among the complications after operation are secondary hemorrhage, infection, and bladder disturbances.

Secondary hemorrhage occurred in 2 cases, in 1 of which it took place from the artery of the clitoris. In a case reported by Franke, in which the posterior vaginal wall was involved, infection took place through the rectovaginal septum and fatal peritonitis developed. Where the urethral orifice is involved in the growth, a catheter should be left in the bladder. In 1 case, because of cicatrization about the urethral orifice, catheterization had to be kept up for a long time. Incontinence was present in 1 case.

When the disease is well advanced, only palliative measures can be employed. The ulcerated surfaces should be kept clean by daily dressings with antiseptic solutions. The hot sitz bath is especially useful in reducing the surrounding infiltration and the amount of the discharge.

Enough morphine should be given to relieve the pain and keep the patient comfortable. When the patient becomes bed-ridden, the greatest care must be taken to prevent bed-sores.

When the growth is fungoid, even though far advanced, thorough curetting with cauterization of the base will afford considerable relief for a short time. The *x*-ray, which has been shown to bring about complete healing of superficial epitheliomata of the face, has proved of very little value in this region. It was tried in 3 advanced cases; but, although the lesion was reduced from a prominent growth to practically a granular ulcer, the infiltration into the depths

continued unabated. In one case, when this procedure was first employed, the pain was relieved for several hours after, but gradually this effect wore off, and the patient seemed to be as comfortable without as with the treatment.

Result. The longest period that a patient has been found to be entirely free from the disease is five years. This patient could not be pronounced cured, for, in a case reported by Schwarz, Olshausen excised a growth which had recurred six years after the patient had been operated upon by Küstner. Zweifel, in the *Cyclopedia of Obstetrics and Gynecology*, vol. xii. p. 311, cites another case in which a recurrence took place five years after the patient had been operated upon by Schroeder. It is evident that statistics showing freedom from recurrence for a shorter time than this are valueless, as far as permanent cures are concerned, and in the absence of any cases that have been free for more than six years, it must be admitted that the disease probably always recurs.

The following cases were free from recurrence for over three years, and 10 additional cases for more than one year:

Author.	Years.	Months.
Rupprecht	5	0
Martin[35]	5	0
Schwarz	4	5
Schwarz	4	5
Schwarz	4	1
Manley[34]	4	0
Schwarz	3	2
Schwarz	3	1
Zweifel[61]	3	0
Kelly	3	0

Schwarz had 2 cases, in 1 of which the patient died of emphysema, and in the other of erysipelas; they had been for four years and seven months and four years and four months, respectively, free from recurrence.

Martin[35] reports another interesting case in which, although the whole of the outer genitalia and one-third of the vagina had been removed, the patient went through a normal delivery two years afterward. The recurrence may take place either in the glands, on the vulva, or in both situations at the same time. Where the vulval growth alone was removed the recurrence was found in the glands in 12 cases, on the vulva in 9 cases, and in both simultaneously in 11 cases. Where the glands were removed from one or both sides, together with the primary growth, recurrence took place in the glands in 6 cases, on the vulva in 9 cases, and in both vulva and the glands in 7 cases. It is therefore necessary to remove the glands and the vulva as widely as possible in the endeavor to prevent recurrence. If, however, there is some contraindication to the more radical operation, it should be borne in mind that a wide excision of the growth will often yield several years of freedom from the disease. Nor should it be forgotten that, as in carcinoma elsewhere, the

earlier the patient is submitted to operation the better her chance of having several years of freedom from a recurrence, even if permanent cure is impossible.

The most frequent cause of death is exhaustion, due to recurrence with ulceration. Five patients died from metastases in the viscera; 3 from hemorrhage from the femoral vein (Schwarz, Leech, and our own Case II.). Three patients died from other causes. In 2 other cases infection took place from the ulcerated tumor, and the patient died of peritonitis.

CONCLUSIONS. 1. Malignant growths of the external genitalia in women are rare. Carcinoma of the vulva occurs in approximately 0.22 per cent. of gynecological patients, and in 5.66 per cent. of patients suffering from carcinoma of the genital apparatus.

2. In a series of 135 cases, the records of which have been carefully examined, the age of the youngest patient was twenty years. The greatest frequency is between the ages of sixty-one and seventy years.

3. Mechanical injuries to the external genitalia are generally quoted as among the principal causes of carcinoma vulvæ. Their etiological relation to this disease, however, is by no means generally evident. On the other hand, long-continued irritation, as, for instance, that caused by a pruritus, undoubtedly has often to do with malignant growths of the vulva. Leucoplakia also has often been noted as a precursor of epithelioma vulvæ.

4. The disease usually begins on the labium majus, and is seen more commonly on the right side.

5. There are two types of the disease—vegetating and infiltrating—which may co-exist.

6. There are four histological classes: scirrhous and medullary carcinoma, cancroid, and melanocarcinoma.

7. The most common symptom is itching. When ulceration has taken place there is usually pain, more or less foul discharge, and some bleeding. Occasionally there are profuse hemorrhages. Intractable insomnia often results from the itching and pain. Dysuria is not very uncommon.

8. Four stages are described by Maurel: (*a*) The pretumoral stage, characterized by an intolerable pruritus. (*b*) The second stage, in which a tumor with infiltration is present. (*c*) The period of ulceration, usually with speedy involvement of the inguinal glands. (*d*) In the final stage ulceration becomes more rapid. Occasionally metastases occurs in the other viscera, and the patient dies of exhaustion The average duration without operation is about two years after the discovery of the tumor; not a few cases, however, last much longer.

9. When the diagnosis is doubtful, the microscopic examination will prove conclusive. Rapid growth, with cachexia and implication of the inguinal glands, is very suggestive.

10. The prognosis is bad. Return—so far as we know at present—is almost inevitable. No case is on record in which the patient remained free from a recurrence for over six years.

11. The best treatment consists in early excision of the vulva, with extensive dissection of the inguinal glands on both sides.

12. The *x*-rays are of little value in these cases. Occasionally they may alleviate pain and inhibit the external growth, but they exert no influence upon the deeper extensions. In non-operable cases the treatment is similar to that emplyed for extensive carcinomatous growths elsewhere.

I wish to express my thanks to Dr. Hunter Robb, Dr. Dudley P. Allen, and Dr. William H. Weir for the use of their cases and valuable assistance in preparing this paper.

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A CASE OF NECROTIC STOMATITIS FOLLOWING MEASLES AND PNEUMONIA.[1]

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The history of the case is as follows: H. A. R., male, aged twenty-one months. The mother states the child has never had any illness. Parents and older brother are well.

February 18, 1904. The *present illness* began eleven days ago with measles. No physician was in attendance, the child's father being a medical student. The mother states that the rash appeared first on the face, and that there was some conjunctivitis; that the child had very little fever and was up and about.

On examination the child was found to be well nourished; the face red and puffy; the eyes inflamed and swollen; no cyanosis. Evidence of rash in the last stage of desquamation on the chest; respirations rapid; pulse 140. Over the right base rales and bronchial breathing are heard.

The next three days were marked by a gradual improvement. On the fourth day the mother reported that she had discovered a patch on the inside of the mouth the night before. On examination a slough was found one and one-half inches long by one-quarter inch wide, running back from the left angle of the mouth, and confined to the inner surface of the cheek. The gums were not involved. The slough was white in color and very dense. There was no odor, although the mother said she had noticed a bad odor the day before. The cheek was slightly indurated, but not edematous; there was a small purplish spot on the outside, opposite the

[1] Read before the Philadelphia Pediatric Society, March 14, 1905.

necrotic area. A mouth wash of hydrogen dioxide, half strength, was ordered to be used at short intervals.

On the 24th the mother requested a consultation with Dr. J. P. C. Griffith, who was unable to see the case until the afternoon. Dr. Griffith, however, concurred when cauterization of the necrotic area was suggested, and this was done at noon on the same day with nitric acid, by Dr. J. M. Spellissy. This cauterization did not appear to be very destructive. In the afternoon, when the child was seen by Dr. Griffith, there had been no spreading of the slough, and the condition of the lungs was found to be favorable. He again agreed to the advisability of more radical cauterization with the actual cautery. At this time no spot was seen on the cheek, and there was no odor. The fauces and tonsils presented a clean appearance. On the next day at noon Dr. Spellissy curetted the necrotic area and cauterized it with the Paquelin cautery; the slough had begun to separate at the centre, but had spread slightly at the periphery. A point on the gums opposite the slough was found to be affected. The cheek was less hard and there was no red spot. Both cauterizations were performed under chloroform anæsthesia. At this visit the pulmonary condition was distinctly improved, although the temperature was still $101\frac{3}{5}^{\circ}$ and the respirations 44. The child was seen again in the evening, and was then found asleep and in a very fair condition.

After that the patient came under the care of Dr. M. G. Tull, and recovered within a few days. I saw the child some three months later, when it was quite well.

Before the necrotic area was cauterized, culture tubes were inoculated with some of the material with the following bacteriological result: "Diphtheria organisms found in both throat and mouth; all the cultures showed, in addition, staphylococcus albus and streptococcus pyogenes; no bacilli nor higher fungi observed."

The diagnosis of this case is open to some question. In view of the presence of measles complicated by pneumonia—one of the commonest causes of noma—the evident tendency of the lesion to spread both on the cheek and to the opposed gums, and the character of the diseased tissue, which was dense, tough, and not like a false membrane, I believed I had to deal with a very early stage of the process that terminates in gangrenous stomatitis. The presence of the Klebs-Loeffler bacillus is in favor of this diagnosis, as the micro-organism has been found in a number of cases of noma, although it probably is not the cause of the disease. Walsh found the diphtheria bacillus in eight cases of noma that he examined, five of them following measles; while in fifteen cases of ulcerative stomatitis the bacillus was not present. Sailer has reported two cases following typhoid fever, in both of which cultures of the diphtheria bacillus were obtained, although it should be mentioned that in one of these there was a history of diphtheria a year pre-

viously. Similar findings have been reported by Freymuth and Petruschky, v. Ranke, Perthes, and others. Animal experiments, when any were made, were inconclusive, and some of the observers expressed the opinion that if the bacillus found were really the diphtheria bacillus its virulence was diminished. Blumer and McFarlane, who studied an epidemic and reported their results in 1901, believe that the disease is due to a thread-like organism of the leptothrix type that does not grow on ordinary media, thus confirming the observations of Perthes in 1899. They exclude the diphtheria bacillus as a causal factor. Apparently the leptothrix referred to was not found by the bacteriologist of the Philadelphia Clinical Laboratory, where the examination of this case was made.

Antitoxin has been given in some of the cases in which the diphtheria bacillus has been found, but the evidence of its usefulness is not clear, and since it appears to be proved that the Klebs-Loeffler bacillus is not the cause of the disease, the use of antitoxin does not seem to be clearly indicated. On the other hand, it is difficult to say whether the active cauterization and curettement employed in this case had anything to do with the favorable termination. Most authorities, however, agree as to the advisability of removing the diseased area as completely as possible, either with with the cautery or with the knife.

THE NEGATIVE VALUE OF KERNIG'S SIGN.[1]

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of Philadelphia.

My purpose in discussing the flexion-contracture symptom, commonly known as Kernig's sign, is not so much one of a desire to question its regularity of occurrence in meningitis as to demonstrate or to disprove at first hand, and as nearly as possible, its negative value—viz., its regularity of non-appearance—if there be such a thing, in conditions other than and uncomplicated by meningitis.

Friis, Osler, F. A. Packard, Netter, Chauffard, Sailer, J. E. Miller and others have shown that the sign is usually though not invariably present in cases which leave no doubt as to the presence of meningitis. Tuberculous meningitis would seem more often to fail to present the typical Kernig contracture than the various non-tuberculous forms of the disease. The cases cited by these

[1] Read before the Section of Medicine, College of Physicians, May 7, 1905.

writers, as well as those studied by myself, show that the unavoidable contraction of the flexor muscles on raising to a right angle the leg extended upon the thigh, or the inability of patient and operator to fully extend the leg when the thigh is at right angles with the body, may not appear until late in the disease, that it may occasionally be unilateral, and finally that it may be transient or even absent throughout the course of a typical meningitis.

In each of 3 cases of cerebrospinal meningitis, all probably due to pneumococcus infection, I have recently obtained Kernig's contracture in marked degree, and in each instance it has persisted from the time the case was first observed by me until the present moment, although of the 3 cases, 2 have entirely recovered (symptomatically) and the third is out of both bed and house, though still under the care of Dr. Wm. Campbell Posey for a slight, persistent optic neuritis. Two of these patients on being examined for the flexion contracture stated voluntarily that they "never could bend their knees straight when sitting up." Both, in fact all 3, still show an angle of flexion of about 110 to 115 degrees, when the thigh is easily carried to a right angle with the trunk. Osler (1887) was one of the first authorities to point out the value of this sign. The most recent edition of his text-book states that "it (Kernig's) is a valuable sign, and has been present in all of our recent cases." He makes no mention of its occurrence in conditions other than meningitis.

Since both of the patients to whom I have referred are unusually intelligent observers in other matters, I am inclined to give them credit for accuracy in their belief as to their inability to fully extend the leg upon the thigh prior to the illness. Moreover, on testing my own limbs for the sign I find that while I can with an effort straighten the leg to the full 180 degrees when sitting erect or when recumbent, none the less it is with considerable discomfort, and I am by no means reluctant to relax the flexor tendons at the proper time.

These facts led me to a study of the cases in the medical wards of the Philadelphia Hospital, only those being selected which showed a complete absence of meningeal symptoms. No case has been considered which has presented even a suspicion of stiffness or rigidity of the cervical muscles, or of delirium, or in which a contracture of the flexor muscles of the leg could be detected when the patient was in the complete recumbent posture. Moreover, Kernig's sign was considered positive only if one or the other leg, when raised by the heel, without an effort that could properly be termed force, presented a distinct flexion angle, varying from 120 to 110 degrees at the knee, when the thigh was at right angles with the trunk.

Miller states that in his study of 190 cases other than meningitis an angle of 180 degrees, or, in other words, an altogether straight

limb, could not be obtained in a single instance, the average angle being 165 degrees.

My own observations do not quite coincide with this finding, a number of cases showing an almost perfect line through the centre of the limb, this being especially true of women as compared with men, and still more frequently of little children. On the other hand, I have already stated my conviction that Miller's assertion is accurate in principle if not in the letter, most healthy persons being unable to avoid a slight flexion angle, unless a considerable degree of force is employed when the test is applied. Of Miller's 190 cases, 45 (23.6 per cent.) gave a positive Kernig sign, he limiting his classification to such instances as presented an angle of not more than 115 degrees. He excludes even one case of acknowledged meningitis which gave an angle of 120 degrees. It will be remembered that Kernig, in his original paper, prescribed the limit for the angle as 135 degrees, a much more generous and probably too lax allowance. Miller's I believe to be too strict a limitation for general use.

As already stated, the total number of cases examined by me was 120, of which 73 were adults and 47 children under the age of ten years. Among the cases studied in adults the following conditions were represented:

	No. cases.		No. cases.
Pulmonary tuberculosis	1	Phlebitis of leg (normal leg examined)	1
Pleural effusion	3	Lead poisoning	1
Cardiac disease	5	Diabetes	1
Cardiac and renal disease	2	Syphilitic bronchopneumonia (conval'ent)	1
Senility	1	Aortic aneurysm	1
Brain lesion	1	Tonsillitis (former convulsions, none recently)	1
Simple anæmia	1	Tonsillitis following typhoid fever	1
Acute rheumatic fever, convalescent (joints entirely well)	9	Cirrhosis of liver	1
Typhoid fever (convalescent)	7	Chronic interstitial nephritis	3
Pneumonia (convalescent)	9	Gastritis	1
Bronchopneumonia and nephritis	1	Emphysema	1
Influenza	8	Apparent health	10
Alcoholism	1		
Eczema	1	Total	73

Among the children there were the following conditions:

	No. cases.		No. cases.
Chicken-pox	1	Epilepsy	1
Pott's disease	1	Trachoma (convalescent)	1
Phthisis pulmonalis	2	Club-foot	1
Gonorrhœal ophthalmia (convalescent)	1	Idiocy	1
Abscess (cervical)	1	Cervical adenitis (not abscess)	1
Otitis media	1	Bronchitis	1
Syphilis (congenital)	1	Gonorrhœa	7
Tetany and convulsions (convalescent)	1	Empyema	1
Rickets	2	Healthy	17
Malnutrition	3		
Eczema	2	Total	47

Of the above conditions Kernig's sign was present in 29 adults in:

	No. cases.		No. cases.
Pulmonary tuberculosis	1	Tonsillitis	1
Pleural effusion	1	Convalescent acute rheumatic fever	3
Convalescent pneumonia	4	Cirrhosis of liver	1
Cardiac and renal disease	2	Influenza	5
Brain lesion	1	Senility	1
Convalescent typhoid	5	Interstitial nephritis	1
Healthy	1	Gastritis	1
Cardiac disease and phlebitis (well leg examined)	1	Total	29

In children Kernig's sign was present in 3 cases:

Rickets	1 case.
Pott's disease	1 "
Gonorrheal vaginitis	1 "

Of the adults the following ages presented Kernig's sign.

Age.	No. cases.	Age.	No. cases.
16	1	47	1
17	1	48	1
20	2	49	1
21	1	50	1
26	1	53	1
28	1	54	1
30	1	57	1
35	1	65	1
36	1	67	1
37	1	73	1
39	2	75	1
41	1	90 Patellar reflexes present. Argyll-Robertson pupil absent.	1
43	1		
46	2		

Of the children the following ages presented Kernig's sign: eight years; one year; three months (case of malnutrition).

Of the total number of patients examined (120 in all), 32 (26.8 per cent.) presented Kernig's sign in the absence, clinically speaking, of meningitis. In 10 of these cases (adult) the patellar reflex was absent, though Kernig's sign was present. In 18 cases (adult) the patellar reflex was absent in the presence of Kernig's sign. Of the 3 children in whom Kernig's sign was present the patellar reflex was present in all.

The Babinski reflex was present in 12 cases: 4 in adults and 8 in children.

Kernig's sign was present in the absence of Babinski's reflex in 22 adults and in 1 child, a case of rickets.

The Kernig and Babinski signs were both present in 4 cases, 1 child (Pott's disease) and 3 adults (1 convalescent typhoid and 2 former pneumonias (both well at the time of examination).

Kernig's and Babinski's signs and the patellar reflexes were all present in only 1 child, a case of Pott's disease.

The Babinski reflex was obtained in the absence of Kernig's

sign in 13 children, 5 of whom were well; also in 2 adults, both of whom were convalescent from typhoid fever.

It may be mentioned that not one of the above cases exhibited the Argyll-Robertson pupil. It is also of interest that many of the smaller children exhibited no plantar reflex.

The patellar reflexes were altogether absent in 17 cases, none of which appeared to be the subject of an evident disease of the nervous system. In 9 of the 17 Kernig's sign was absent and in 8 it was present.

A statistical study such as this suggests many interesting questions, among which not the least puzzling is the connection that may exist between Kernig's sign and a former, perhaps unrecognized, meningitis or encephalitis. In none of this series of cases, however, could such a history be elicited.

Another unsolved query is the cause and significance of Kernig's phenomenon. I must confess that I incline to the theory, already proposed by more than one observer, that it is an exaggeration of a normal tendency to contract on the part of the flexor tendons, due to irritation or disease of the brain or both, and, of course, attended by meningitis.

Certainly the presence of this phenomenon in 23.6 per cent. of the cases in Miller's series, and 26.8 per cent. of my own series, all non-meningitic cases, would seem to indicate that there need be no serious brain or cord involvement present to evoke the sign. The disparity in percentage between the two series of cases is remarkably slight if we consider the difference in the limit set for the flexion angle: in the former 115 degrees and in my series 120 degrees. Shields[1] has also studied a series of 100 non-meningitic cases in which he found Kernig's sign present in only 5 instances—3 unilaterally and 2 bilaterally. Clark[2] reports 3 cases of meningitis (1 epidemic, 2 tuberculous), in all of which Kernig's phenomenon was absent.

It would seem that, as a result of the entire study, one must conclude that Kernig's sign is by no means a positive indication of either meningeal or brain or cord involvement, but that when present in association with other signs of meningitis it may be looked upon as confirmatory to a high degree. Undoubtedly in the majority of cases it persists far into the convalescence, and is usually one of the last signs to disappear. In certain cases it probably remains as a permanent indication of a former undetermined lesion.

[1] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1902. [2] Ibid.

DIMINUTIVE SPLEEN.

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On March 28, 1901, a native Filipino woman, formerly an inmate of the San Lazaro Hospital, Manila, P. I., for leprosy, came to autopsy. Decomposition was well advanced. Body that of a woman about thirty years of age, well built, fairly well nourished, presented no abnormalities, nor any signs of advanced leprosy. On opening thorax and abdomen no abnormalities were noted until a search for the spleen was made. A small spleen, at first taken for an accessory spleen, was seen on the surface of a mass of adipose tissue occupying the space usually filled by the spleen. On more careful search, after removing and cutting the mass of fat into small pieces, no further trace of splenic tissue was found. The spleen was 32 mm. long, 10 mm. wide, and 9 mm. thick at thickest point; and weighed $2\frac{1}{10}$ grams. In shape it simulated a normal organ. Color was a little deeper than the normal, suggesting the color of a malarial spleen; capsule smooth, slightly thickened, and regular; bloodvessels, normal. Organ was firm. One or two subcapsular hemorrhages were noted. Two blocks from opposite sides were removed for histological study. Sections, stained in hæmatoxylin and eosin, showed a slightly thickened capsule, abundant thickened trabeculæ, numerous large arteries, with much thickened and degenerated walls, in which lime was deposited and endothelium thickened. Splenic pulp was much diminished. One Malpighian corpuscle was noted. This was small and mostly composed of connective tissue. The spleen in no way suggests contraction from inflammatory processes as a cause for its size. The retroperitoneal lymphatic glands were large and calcified.

Through the kindness of Dr. N. F. Baker, of Fulton, Mo., I had the opportunity to examine one case of acephalus, full term, in which a small spleen and symmetrical liver, the left lobe filling the left hypochondriac region, were found.

I found, upon inquiry, a scanty literature on diminutive spleen, and few cases reported of absence of spleen. Ziegler says that "the complete absence of the spleen is rare and usually occurs in well-built individuals." Birch-Hirschfeld says: "Absence of spleen is often associated with acephalics and extensive malformation of the abdominal organs." Otto failed to find the spleen in a well-formed five months' fœtus. Birch-Hirschfeld failed to find a spleen in a newly born child, in which the liver was symmetrical and filled the left hypochondriac region. Robert failed to find a spleen in a three-year-old child; in this case the stomach was tubular.

In the case of Arnold there were malformation of the heart, defect in spleen, and enlarged lymph nodes. Rudimentary development of spleen is frequent.

Paulesco found a spleen 5 cm. long, 1 cm. wide, and 26 mm. thick on the surface of a mass of fatty tissue occupying the usual splenic position. No abnormalities or enlarged mesenteric glands were found. Meinhard in 1845 and Koch and Wachsmuth in 1879 report one case each of complete absence of spleen in otherwise normal adults, ages fifty-seven and forty-nine respectively. In 1896 Maximiliano Olaechea reports complete absence of spleen in a fatal case of typhoid. All organs were normal save enlarged mesenteric and bronchial lymphatic glands. Hodenpyl in 1897 reports complete absence of spleen in a male adult thirty-two years old. In this case the mesenteric and retroperitoneal glands were enlarged. Bidner reports a case of absence of spleen in an acephalic with enlarged lymphatic glands in abdomen.

Cases of absence of spleen: Martin, 1; Birch-Hirschfeld, 1; Meinhard, 1; Koch and Wachsmuth, 1; Libby, 1; Ramsey, 1; Olaechea, 1; Hodenpyl, 1; Patillani and Moroni, 1; Arnold, 1; Bidner, 1; Otto, 1; total 12. Doubtless several more cases have been noted, as the absence of spleen in monsters is mentioned as early as 1564, and again by Heusinger in 1820.

Rudimentary spleen: Paulisco, 1.

The literature on atrophic spleens is more extensive and treats largely with conditions secondary to pathological lesions, or to atrophic conditions of the spleen. Judging from the gross and microscopic conditions of the spleen, described above, its diminutive size was not due to contractions from pathological lesions late in life, but to arrest in development, most probably early in embryonic life. In the literature at hand a larger number of cases of complete absence of spleen than diminutive spleens is found.

Clinically, in diseases associated with enlargement of the spleen, the absence of or a diminutive spleen may become of considerable importance. Normally, the upper extremity of the spleen is situated a few centimetres to the left of the body of the tenth thoracic vertebra. The upper and lower borders are parallel to the ninth and eleventh ribs, respectively, as far forward as the midline or anterior axillary line, near which lines the rounded lower extremity is located. About one-third of the upper portion, that portion separated from the thoracic wall by the diaphragm, is covered from above by the lower margin of the left lung. Ordinarily, at the scapular line the lower margin of the lung is at the ninth rib and at the vertebral column as low as the eleventh rib. The line of the lower margin of the lung is nearly horizontal, and consequently forms an acute angle with the line of the upper border of the spleen, which is inclined slightly downward—pulmonosplenic angle. Consequently, on percussion a relatively dull splenic note is elicited in a triangular area

bounded above by the ninth rib, below by the lower border of lung, indicated by a line extending from the articulation of the eleventh rib at vertebral column to the ninth rib at scapula line, and to right by vertebral column. The remainder of the splenic area elicits a dull note, save when mixed with tympany due to a distended stomach or colon. To the right the splenic dulness extends into the dulness of the spinal column. Owing to its position under the diaphragm, enlargement of the spleen with a corresponding enlargement of splenic dulness takes a downward and forward course. In case of dislocation a downward and forward course is usually taken. In these cases the relative splenic dulness may be absent or greatly diminished, and an area of tympany may be found between the upper extremity of spleen and vertebral column. But the outline of the spleen is not necessarily altered.

The literature indicates that in most, if not all, of the cases in which there is no spleen or a very small spleen, the usual splenic space is occupied by an irregular mass of fat, which may not simulate, either in shape or size, the spleen. In the case at hand the mass of fat in which the small spleen was found seemed to be continuous with the omentum, was of a globular shape, much smaller than an ordinary spleen, and was located farther toward the left side than the upper extremity of the spleen. Owing to its consistency the fat should give a percussion note relatively equal to that of the spleen.

In the cases of Koch and Wachsmuth, Paulesco, and Olaechea, typhoid fever was the cause of death, and in each the authors were unable to find the spleen. From their post-mortem records, especially that of Paulesco, and from my case, it occurs to me that the following factors might aid in determining the presence or absence of a spleen in these doubtful cases:

1. If the mass of fat were outlined its shape would be irregular, and other than the oblong or splenic outline; more round.
2. The posterior border would be farther from the vertebral column, leaving possibly a narrow tympanitic area between the mass and the vertebral column.
3. The relative splenic dulness in the lower posterior border of the lung would be absent or moved anterior, and the splenic angle would be materially changed or absent.
4. Traube's similunar space would extend farther posteriorly.
5. The size and shape of the mass of fat would remain practically constant.

In cases of dislocation of the spleen, which usually take a forward and downward direction, the tympanitic area near the vertebral column would be present, but here the usual splenic outline would be found and the edge of the spleen probably could be palpated at the lower margin of the thoracic wall.

In the case under consideration the *flexura coli sinistra* could

dislocate the mass of fat more easily than it sometimes displaces the spleen, to give a tympanitic note over the otherwise dull area. Naturally, if tympany obscured the splenic area from day to day no further data regarding the spleen could be collected.

LITERATURE.

M Gonzaly Olaechea. Caso raro de ausencia del bazo. Cron. méd., Lima, 1896, xiii., 147.

E. Hodenpyl. A Case of Apparent Absence of the Spleen, with General Compensatory Lymphatic Hyperplasia. Proc. New York Path. Soc. (1897-98), 1899, p. 185.

Paulesco. Sur un cas d'arrêt dans le developpement de la rate Journ. de méd. int., Paris, 1898, ii., 114.

Hodenpyl's article collects most of the literature. All of the remaining literature may be found in the Index Catalogue of the Library of the Surgeon-General's Office.

TORSION OF THE GREAT OMENTUM.

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INTRODUCTION. In the first volume of the *Annals of Surgery*, pp. 405–430, published in 1900, Mr. Lawford Knaggs, of Leeds, drew attention to the occurrence of volvulus or torsion of the intestine associated with a hernia. His interest had been drawn to the subject by two cases which occurred in his private practice. Both examples presented well-marked signs and symptoms of the curious condition present, so that it could not be overlooked. In the accounts of other examples which are given it is noteworthy that a very large number were fatal. The evolution of our knowledge of any disease is almost always the same. The severe and fatal forms first attract attention; later, other and milder clinical manifestations are recognized; and, finally, given the necessary circumstances (such as the presence of a hernia, as in this example), the condition is recognized as occurring frequently. Mr. Lawford Knaggs' paper is really an introduction of the subject from the period of rarity to the middle stage. As yet it has not advanced to the third or "frequent" stage. Though only writing on a kindred subject, torsion of the omentum and not of the bowel, we must express our opinion that volvulus is a very common consequence of the strangulation of a hernia. When the strangulation is at the hernial orifice, this becomes the fixed point about which the protruded loop of bowel twists. The twisting agents are, firstly, the meteorism in the strangulated coil; and, secondly, the movements of flexion of the thigh.

If the strangulation is not tight, the point on which the bowel has twisted may be carried within the abdomen by the force of the twist, when the intestinal obstruction becomes intra-abdominal. Should the hernia alone be operated on, and the volvulus not be reduced spontaneously, as will occur very easily when the twisted coil is set free from the hernia, the patient will die of unrelieved obstruction. These cases are rare, and naturally will continue so, because in strangulation of the bowel the volvulus is secondary and makes a previously slight obstruction absolute. As an example of this, reference is made to a case recorded in the Erasmus Wilson *Lectures*, 1904, pp. 58–60, in which a volvulus was noted. It is a matter of common surgical knowledge that in the strangulation of a hernia the situation of the obstruction is at the hernial orifice. If there is torsion of the bowel it will probably be intrahernial, and very rarely intra-abdominal. The volvulus is undone after the reduction of the bowel by the presence of the gas within it. But if surgeons will take the trouble to look carefully, volvulus of the gut within the hernia will be found a common, if not a very common, occurrence, but it is of little or no practical import—just as the torsion, which can be seen accompanying almost every internal form of intestinal obstruction if looked for, requires no special treatment. Torsion of the bowel in strangulated, obstructed, or painful and irreducible herniæ is far from an uncommon event. What, then, with regard to the omentum which is often alone or sometimes accompanies the bowel? Before entering upon the subject-matter of this paper reference must be made to another structure, besides bowel and omentum, which descends through the inguinal canal into the scrotum. This organ is the testis. The twists found in structures which have passed through this region are formed slowly by the addition of frequent small increments. The acute volvulus of the bowel in strangulated herniæ is an exception. Normally, the testis is in the scrotum by the eighth or ninth month of intrauterine life. Imperfectly descended testes accompany the majority of the recognized cases of torsion of the cord, and are in the situation to become twisted by the movements of the trunk and legs. A careful examination of imperfectly descended testes showed that small degrees of torsion, half a complete turn, were commonly found in cases which had suffered from sudden and sharp attacks of pain in the gland. Attacks of pain in an imperfectly descended testis were given a marked significance by this fact.

Both bowel and imperfectly descended testes in the inguinal region are often twisted. We should expect the omentum which is exposed to similar conditions when in a hernia, to suffer likewise. With the object of introducing this, and of collecting all that is known on the subject within reasonable compass and in an accessible form, the authors have written this paper. No cases beyond the end of 1904 have been included.

HISTORY. The first example of torsion of the omentum, described by Oberst in the year 1882, was found in the sac of a painful irreducible inguinal hernia of old standing in a man aged thirty-five years. From this date to the year 1893, when Demons described an example, no other case was recorded. In 1898 3 new examples were added, 1 being added by each of the following: Bayer, Eiselberg, and Monod; 10 more were published in 1900; 6 more in 1901, and 5 more in 1902. In 1903 a large number of new instances were described, 16 in number. Up to the end of 1904 only 7 more have been added. Four new ones are recorded in this paper, thus bringing the total number up to 53. The fact that the first instance was described in 1882 cannot be urged in favor of a new pathological condition occurring then for the first time. Neither can the fact that no cases are described in 1890 and 16 in 1903 be attributed to such a condition as that represented by the phrase "good and bad harvests." The most intelligible explanation of the history of the disease is that, although it had occurred for centuries and perhaps thousands of years, the condition was first *observed* by Oberst in 1882. For the next eleven years it continued to be overlooked, until in 1893 Demons rediscovered it. The seeds sown by these authors did not bear fruit until 1898; and though 1899 was barren in this regard, the next year bore full fruit, 10 cases. Since then surgeons have begun to notice the condition, and by the end of 1904 53 cases in all are known.

A further point is of interest—viz., that Lucas-Championnière (observations 15 and 17) was the first to report more than 1 case. Yet it cannot be held that he is the only man who up to 1901 had seen 2 cases. To him must be given the credit of being the first to *recognize* 2 of his own. Nordman (observations 28 and 29) in 1903 reported 2 cases, and Rudolph in the same year reported 4 others, and to him belongs the credit of observing the largest number of cases and publishing the longest contribution to the subject (observations 30 to 33). Mauclaire in 1904 published 2 cases (46 and 47), and the authors of this paper now add 4 more (50 to 53).

History teaches us, also, that only the most obvious cases were recognized. And although we know that intra-abdominal torsion of the omentum must be infrequent, torsion of it within the sac of a strangulated or painful and irreducible epiplocele is a fairly common event. Both the history of the reported cases and our own observations bear out this suggestion. To exemplify how these cases may have been mistaken or overlooked we have included a section on the Inferential Diagnosis.

CLINICAL CLASSIFICATION. The cases of torsion of the omentum naturally fall into three main clinical groups:

1. *Abdominal.* In this type there is no hernia present and the torsion is purely abdominal. There are 6 examples: Baldwin (22),

Syme (23), Stewart (43), Noble (45), Scudder (48), Corner and Pinches (51).

2. *Hernial.* The torsion occurs solely within the sac. There are 6 examples: Oberst (1), Heinlein (26), Rudolph (31), Capette (38), Corner and Pinches (52 and 53).

3. *Hernial and Abdominal.* The torsion is not limited to the hernial sac, but extends into the abdomen, or there may be a series of twists in each. The remaining 41 cases belong to this variety. Two subdivisions may be recognized:

1. When a hernial tumor is present, 30 cases.

2. When the hernial sac is empty the usual contents being reduced into the abdomen, 11 cases.

Cases of the second class, the hernial, are the ones most easily overlooked and will be found, we believe, to be the most frequent.

Clinical Diagnosis Made in the Reported Cases. 1. *Abdominal.* Of these 6 cases, 5 were diagnosticated as appendicitis, probably accompanied by a local abscess; the other as a suppurating omental hydatid.

2. *Hernial.* Strangulated hernia was the diagnosis made in 4 cases, irreducible hernia in 1, and incarcerated hernia in the other.

3. *Hernial and Abdominal.* In this large group the following diagnoses were made:

Strangulated inguinal hernia	14 cases.
Incarcerated " "	1 case.
Irreducible " "	2 cases.
"Inguinal hernia"? condition	2 "
Strangulated umbilical hernia	1 case.
Reduction of hernia *en masse*	2 cases.
Sarcoma of testicle	1 case.
Appendicitis	9 cases.
Intestinal obstruction	1 case.
Abdominal tumor	1 "
Intraperitoneal abscess	1 "
Chronic peritonitis	1 "
Torsion of omentum	1 "

In 4 cases no diagnosis was made. The diagnosis of torsion of the omentum was made once, by Rudolph (32).

Clinical Etiology. *Sex.* Torsion of the omentum occurs more frequently in males than in females. Of the 53 recorded cases, 36 were males, 17 females. Males, 67.92 per cent.; females, 32.07 per cent.

During the years 1898–1902 inclusively, the number of cases of inguinal hernia admitted to St. Thomas' Hospital was 1080; of these 951 were males, 129 females; about 8 to 1. In this same period there were 191 strangulated inguinal herniæ, of which 184 were males, 7 females; about 26 to 1. The presence of a strangulated inguinal hernia in a woman should raise the suspicion of the presence of a uterine appendage or twisted omentum in the sac.

Age.	No. of cases.		
0 to 10 years	0		
11 " 20 "	1	=	1.88 per cent.
21 " 30 "	5	=	9.4 "
31 " 40 "	14	=	26.4 "
41 " 50 "	20	=	37.7 "
61 " 70 "	0		
71 " 80 "	1	=	1.88 "
Not recorded	3	=	5.6 "

The most frequent period for the torsion to occur is middle life, from thirty-five to fifty-five years of age; 39 cases, 78 per cent., occurred during that period. The youngest recorded example was seventeen (Chavannaz, 10). The oldest was seventy-nine (Wiener, 9).

Hernia. With the exception of the 6 purely abdominal cases, a hernia was present in every instance, 90 per cent. The various forms of herniæ present were as follows:

Right inguinal	33 cases	=	70.2 per cent.
Left "	9 "	=	19.1 "
? side "	3 "	=	6.38 "
Ventral	1 case	=	2.1 "
Umbilical	1 "	=	2.1 "

In no case was there a femoral hernia present.

These ruptures had been previously reducible, with the exception of 3: L. Championnière and Mauban (17), Weisinger (16), Heitz and Bender (11), Makins (50).

In 4 cases the hernia had been present between one and five years, 5 cases between six and ten years, 8 cases between eleven and fifteen years, 4 cases between sixteen and twenty years, and 6 cases between twenty-one and thirty years. The hernia is commonly one of long standing, 33 per cent. having been present under ten years, 45 per cent. between ten and twenty years, and 22 per cent. between twenty and thirty years.

The Previous History of the Cases. In only 16 of these instances is there any reference to a previous illness at all referable to the omental torsion. Of these 16, 9 occurred in 1903 and 1904; so that it is possible, nay, probable, that the previous history had been overlooked. The past histories in 12 of these cases are as follows:

Case 1. Wiener (9). Four weeks before the acute attack the patient had a sudden attack of severe pain in the right iliac region, which lasted twenty-four hours and then subsided.

Case 2. Nordmann (28). For several weeks the patient had suffered from gastric disturbances accompanied by abdominal pain.

Case 3. Malherbe (41). For two years before the operation the patient suffered from attacks of pain in the right inguinal region, with vomiting. During this period there were five attacks, each terminating when the hernia was reduced.

Case 4. Vignard (40). For nine years before the operation there were attacks of pain in the right side of the abdomen, which lasted four to five days. There were four or five attacks each year.

Case 6. Audier (44). There had been several attacks resembling intestinal obstruction.

Case 7. Noble (45). For two years before operation, at irregular intervals, the patient had attacks of sudden pain in the abdomen, attended by nausea and vomiting.

Case 8. Corner and Pinches (51). History of several attacks of pain and discomfort in the right side of the abdomen, not severe enough to cause the patient to take to her bed.

Case 9 and 10. Corner and Pinches (52 and 53). Both men had had attacks of pain in their herniæ, but none so severe as those for which they were operated on.

Case 11. Trinkler (49). There was a history of previous attacks of abdominal pain.

Case 12. Makins (50). She had suffered from several attacks of pain in the hernia, sometimes accompanied by sickness.

The other examples are recorded by Hochenegg (8), Championnière (15), Quenu (29), and Morrison (39).

It is most important that the significance of previous crises or attacks of pain in the hernia or in the abdomen should be recognized. These may have been produced by colic, appendicitis, or some such condition, quite unconnected with the omental twists. But many twists, sometimes ten, are frequently found. These have been formed by slow increments of the torsion. It is tempting and not irrational to refer the previous attacks of pain to some of the increments being greater or more forcible than the others. This is borne out with regard to the testis; for instance, the more acute increase of torsion, which caused the attack of pain, has been untwisted, giving perfect relief. A similar suggestion with regard to the previous history has been strongly urged in connection with volvulus of the cæcum in a recent paper.[1] Future observers will gain much in paying more attention to the history of cases of this nature.

If this surmise is correct—that these attacks, or some of them, are produced by increased twists, although only the more severe cases are yet recognized—the previous history of the patients illustrates the clinical history of milder examples, due to subacute and chronic torsion of the omentum and hitherto unrecognized. It may be expected confidently that the observations in future will tend to make the occurrence of omental torsion more frequent, just as in the cases of torsions of the bowel and of the testis.

Clinical Symptoms. *Onset.* In only a few of these cases is any reference made to the onset of symptoms. In 21 cases, 37.7 per cent.,

[1] Corner and Sargent. St. Thomas' Hospital Reports, 1903, pp. 407–420; and Annals of Surgery, January, 1905.

the onset was sudden, and in seven, 13.2 per cent., gradual. In the purely abdominal cases sudden onset was noted four times, gradual once. Below are given some examples of the method of onset.

Monod (5). Immediately after an effort, the patient felt a sharp pain in the right iliac fossa, the pain being likened to a cut with a knife.

Noble (45). Sudden onset with excruciating pain in the abdomen, attended by nausea and vomiting.

Malherbe (41). Four hours after a meal sudden violent pain in the right iliac fossa, most intense at McBurney's point.

Corner and Pinches (51). The patient had been ill for ten days with pain in the right side.

Broca and Chavannaz (10). Suddenly one morning an irreducible tumor appeared in the right inguinal region, without any signs of strangulation being present.

Scudder (48). Sudden onset with abdominal pain and vomiting.

Pain. Pain is the most constant of all the symptoms, and in nearly all the cases where any details are given pain is said to be present, and is one of the first symptoms to appear. In investigating this symptom it will be best to divide the cases into three types:

1. *Abdominal.* Pain in the abdomen was present in all these cases; in 4 it was situated in the right iliac fossa, and in one it is said to have been diffuse but most intense at McBurney's point.
2. *Hernial.* In 4 cases pain is noted as being present in the hernia.
3. *Abdominal and Hernial.*

TABLE SHOWING THE SITE TO WHICH THE PAIN WAS REFERRED IN THESE CASES.

Pain referred to hernia alone	14 cases
" " abdomen alone	17 "
" " both	7 "
Pain not mentioned in any particular region	3 "

In 11 cases the pain is especially referred to the right iliac fossa, and in 1 is said to be most intense at McBurney's point. As a rule it is very acute and persistent, but in 1 case (Morrison, 39) there were exacerbations of pain from time to time. In most cases it was sufficiently severe to cause the patient to take to his bed.

Vomiting does not appear to be a very constant symptom; in 19 cases, 35.8 per cent., it is said to have been present; in 14, 26.4 per cent., the absence of vomiting is noted; and in 20, 37.7 per cent., no mention of vomiting is made. Where it has been noted it is usually frequent and violent. In 1 case (Lejars) it is noted that the vomited matter was greenish. Nausea without vomiting was noted in 9.4 per cent of cases.

Bowels. Condition of the Bowels. Diarrhœa in 2 cases; bowels open in 16 cases; bowels inactive in 11 cases; flatus alone passed in 5 cases.

From the above table it will be seen that the most commonly recorded condition is that the bowels were open; but this term is a vague one. In the majority of cases it is not stated on what day of the disease they were opened, or how often. Absolute "obstruction" is noted five times and the passage of flatus alone in 5 instances. On the whole the condition of the bowels seems very variable, but the most common condition is one of inactivity.

Pulse. As a rule the pulse is accelerated, varying from 80 up to 120 per minute.

Temperature. The temperature is usually slightly raised; in 21 cases, 39.6 per cent., it is noted that the temperature was above normal; in only 3 is it said to have unchanged. The highest recorded temperature is 101.8°; the usual is between 100.4° and 101.4°. In no case is it said to have been subnormal.

CLINICAL SIGNS. *Tumor.* In all but 3 of these patients a tumor could be felt on examination, but its situation varied.

1. *Abdominal.* In 4 of these a mass could be felt in the right iliac fossa; in the other 2 there was tenderness and rigidity in this region, but no definite tumor could be detected on palpation.

2. *Hernial.* All of these patients had a hard, painful, irreducible swelling in the inguinal region.

3. *Hernial and Abdominal.* The following summary shows the tumor present in this series:

Situation of Tumor: A hernial tumor alone present, 12 cases; an abdominal tumor alone present, 7 cases; tumors present in both situations, 19 cases; neither tumor detected, 3 cases.

From this it will be seen that it is most common for a tumor to be present both in the hernia and in the abdomen, and it is very frequently noted that the hernial and abdominal tumors appear to be continuous.

The tumor is, as a rule, hard, tender on palpation, dull to light rather than to heavy percussion, and is often said to have had an irregular surface. Dulness was noted nineteen times in this series. General distention of the abdomen is not a common sign; it was only noted seven times, 13.2 per cent.

PROGNOSIS. All of the cases recovered except 8. The fatal ones were recorded by Eiselberg (4), Monod (5), Heitz and Bender (11), Lejars (14), Moresco (25), Tremolière (34), Zeller (37) and Audier (44). Of these Tremolière's was too bad for anything to be done; Audier's was operated on as a desperate measure; Monod's and Heitz and Bender's died of pneumonia, and Lejars' of delirium tremens. Zeller's record is so short that nothing can be said. Eiselberg's case died thirty-eight hours after operation, presumably of peritonitis. These results show that when submitted early to operation the prognosis is good.

The mortality for these 53 cases, in which the majority of observers recognized the pathological condition of torsion of the omentum

for the first time, is 13.2 per cent. But if the cases in which the fatal result was not concerned with the surgeon are excluded, the percentage mortality is 4.1; there are very few conditions which can show so small a mortality in the first chapter of their history.

INFERENTIAL DIAGNOSIS. All diagnosis is inferential: the conclusion is arrived at by means of inferences drawn from observations. These inferences can only be deduced correctly when born of sound knowledge. From the previous part of this paper practically all that is known of torsion of the omentum can be learned. It was decided, therefore, to include a section on the inferential diagnosis, in which we will draw inferences from the cases of others and from our own. As a new disease was not discovered in the first case of omental torsion which was reported, but merely an old one recognized, this section will be interesting and instructive in affording examples in which the twists were or may have been overlooked. Further, some authors have included under the head of omental torsion cases which, after mature consideration and study of the records of all available examples, we do not feel justified in accepting on the evidence adduced. The first three and the fifth instance to be quoted are examples of this.

To begin with, we will briefly summarize the conditions which should lead to a suspicion of the existence of torsion of the omentum. The subject is most commonly a man of middle age, with an inguinal hernia of some years' standing, which has become troublesome and gives rise to certain symptoms which can be summed up as those of subacute intestinal obstruction. Vomiting may be absent and both feces and flatus may be passed. On examination the hernia will most probably appear to be painful and irreducible, incarcerated or strangulated. Usually a tumor can be felt both in the scrotum and on the right side of the abdomen. Should such a condition be found in the inguinal hernia of an adult woman, it will either contain an ovary and Fallopian tube or twisted omentum. Occasionally only the abdominal tumor is present, the sac appearing to be empty. Such is the clinical picture in brief, which should raise suspicions of omental torsion.

It has been stated that to Lucas-Championnière belongs the honor of being the first man to recognize two cases of torsion of the omentum. These were published separately in 1900 and 1901. In 1898, previous to his having recognized the condition, he published a case which is briefly reproduced.

Lucas-Championnière[1] Male, aged fifty-three years. Right inguinal hernia since infancy; had worn a truss. For some time past the hernia had become more painful. On examination there was a large irreducible inguinal hernia, tender on pressure; the abdomen was hard and tender. The bowels were open. Operation:

[1] Bull Soc chir, 1898, pp 195–197.

The inguinal canal was occupied by an enormous cord continuous with a tumor in the scrotum, which was very adherent to the sac. In the centre was a little black liquid, apparently altered blood; the mass was prolonged upward into the abdomen, where it joined an enormous tumor which occupied all the anterior part of the abdominal cavity and descended into the pelvis. At the upper part it was in juxtaposition with the transverse colon and was adherent to the anterior abdominal wall. This tumor was thick, hard, lardaceous, and consisted of the entire omentum. Recovery.

This case agrees with everything which has been said of omental torsion, with the exception that the torsion must have been very slow and subacute to account for the great size of the omentum and the many adhesions which it had contracted.

Potherat in 1900, previous to his recognition of the pathological aspect of torsion of the omentum, published a case which he says was like that recorded by Championnière in 1898, which we have just reproduced. The following is a brief summary of Potherat's case.

Potherat.[1] Male, aged sixteen years. Right inguinal hernia. When first seen there was a hard, rounded tumor, obscuring the testicle; above it was a hard, irregular, thickened cord entering the inguinal canal. The patient had had slight symptoms for some months, was unable to walk, and had suffered from loss of appetite and flesh. Diagnosis: neoplasm of testicle and epididymis. Operation: Castration was performed and the cord resected high up in the canal. On examining the specimen it was found that it consisted of a mass of thickened, heaped-up omentum, which surrounded and buried the testicle; it was dark in color and resembled that described by Championnière (1898). The condition was thought to be due to inflammatory lesions affecting an old omental hernia.

Like Championnière's, this seems to have been a case of chronic torsion of the omentum; but, unlike his, the trouble appeared to be confined to the testis.

Verdelet and Rocher recorded a case in 1900 in which a hernia became irreducible, and at the operation there was found a condition in which torsion could very easily have occurred.

Verdelet and Rocher.[2] Male, aged fifty-six years. Reducible right inguinal hernia for forty years. The patient wore a truss. The hernia suddenly became irreducible without any symptoms being present; it remained irreducible for six months and was then replaced. Three weeks later, after a fit of coughing, it again became irreducible. After two days, during which taxis was repeatedly tried, the hernia was partially reduced. In the right side of the scrotum there was a soft lobulated mass, with a pedicle running up

[1] Bull. Soc. Chir., 1900, pp. 525-531.

[2] Journ. de méd. de Bordeaux, September, 1900, pp. 559-560; Gaz. hebdom. des scien. méd., June, 1900, pp. 291-293.

above Poupart's ligament. The tumor was not painful; it was dull on percussion, except in the upper part, where it was a little resonant. All had been reduced except a rounded mass the size of an orange, which was irregular, hard, and lobulated, but not painful on pressure. From its upper part there was a pedicle going in the direction of the inguinal canal. It was movable in the scrotum and situated behind the hernial mass. Operation: In the sac a large, fat, vascular mass was found, behind which was an irregular, lobulated, fatty tumor the size of a fetal head. There was a pedicle of the same nature, with large vessels in it, running toward the inguinal canal. There was also a right hydrocele present. The tumor was formed by the "rolling up" of the omentum on itself.

They describe the tumor as consisting of "rolled-up" omentum. That rolling up is not the same as twisting has not been recognized by all observers, some of whom have taken this case to be one of proved torsion. Had other symptoms than mere painless irreducibility of the hernia been present it would have been probable that torsion of the stalk above this "rolled-up" omentum had taken place. As it is, there is no evidence that torsion did occur, though it is hard to realize that it did not.

In 1902 Spellissy, in a paper on the diagnosis of appendicitis, reported a most suggestive case. Though a hernial sac was present all the signs and symptoms were intra-abdominal.

Spellissy:[1] Female, aged forty years. Right inguinal hernia for eight years. History of one day's illness, accompanied by absolute inactivity of bowels and vomiting. Diagnosis: Appendicitis. Operation: In the abdomen was found a large mass of omentum weighing about two pounds, which was in a gangrenous condition. No abscess was observed and no torsion is mentioned. The patient died on the fourth day.

This case leads up to another American one in which the symptoms were abdominal and no hernial sac was present. Although the account is not clear, some authors have included Eitel's case as one of omental torsion—*e. g.*, Scudder (48) and Noble, (45).

Eitel:[2] Male, aged forty-four years. Patient noticed the abdomen was of large size. It was tapped and three gallons of fluid drawn off. Twenty-four days later it had again become distended and two and one-half gallons were drawn off. After tapping a tumor was felt in the hypogastrium, seven inches long, four broad, and four thick, soft, movable, and neither tender nor painful on examination. Operation three days later: An omental tumor was found, rolled inward and upward, producing constriction and partial obstruction to the return circulation in it. Its veins were enormously distended. It was unravelled without difficulty and the omentum spread out; whereupon the dilatation of the veins

[1] Annals of Surgery, 1902, vol. i, p. 760.
[2] New York Medical Record, May, 1899. (Reference not confirmed.)

disappeared and it became normal in appearance. The patient had been in the habit of carrying a heavy box which pressed upward and inward just below the umbilicus. No torsion above the tumor formed by the rolling up of the omentum was present.

Again, there is the word "rolled," which renders the diagnosis of torsion difficult. This, coupled with the fact that other clinical data, such as the condition of the liver, are wanting, plus the knowledge that no other case of known omental torsion has been accompanied by such ascites, makes it unwarrantable for us to include this case.

Now, to draw attention to 4 more cases, 1 of Mr. Stephen Paget and 3 of our own. By these examples we wish to indicate the kind of case in which it seems probable that some twisting of the irreducible omentum is the cause of the pain and of the other symptoms. Any surgeon can realize how very easy it is to overlook such a turn buried deep at the root of a large omental hernia; and, owing to the absence of landmarks on the omentum itself, it is impossible to recognize even the straight position much less the slightly twisted one. The dilated condition of the veins makes it obvious that there is some pressure obstructing the blood flow in them. If this is not caused by inguinal rings it must be brought about by some change in the omentum itself. Under these circumstances torsion seems to be the most plausible explanation. Moreover, we have observed it in two cases which were very like the one just quoted.

Stephen Paget. In the College of Surgeons Museum there is a specimen (2621, A) of omentum removed in a case of supposed strangulated hernia. The following account is taken from the notes furnished with the specimen.

Male, aged sixty-three years. Right inguinal hernia for thirty-eight years. A truss had been worn. Recently the hernia came down and he was unable to replace it. Half of the tumor was returned by taxis, but a portion remained and gave a "crumbling" sensation to the hand suggestive of blood clot. The hernia increased in size. The bowels were opened; temperature normal; no vomiting; no pain. Operation: There was a thick sac containing omentum. The constriction shown in the specimen had no connection with the external ring. There was a band of omentum, arising below the constriction, adherent to the bottom of the sac. The lower half of the tumor was deep red in color.

Corner and Pinches (Case 1). W. A., male, aged twenty-five years, was admitted to St. Thomas' Hospital for a painful irreducible inguinal hernia on the left side. At the operation it was noticed that the sac contained omentum only, with some fluid; also, the omentum, though swollen, was not strangulated, and could be reduced by steady pressure without needing a "herniotomy." There was no evidence that the fluid came from the abdomen; indeed, the swollen omentum would seem to have acted as a cork

and prevented it. If so, the fluid formation must have been local, in the hernia sac. Considering the extreme ease with which such a condition is overlooked during the manipulations of the operation, the case may very well have been one of torsion of the omentum.

Corner and Pinches (Case 2). W. P., male, aged forty-six years, was admitted to St. Thomas' Hospital with a painful, irreducible, right inguinal hernia. There was no vomiting. The rupture was very large. At the operation it was found to consist of an enormous mass of omentum with distended veins. More omentum was withdrawn from the abdomen, the stalk ligatured, and the mass removed. No torsion was noticed, as it was not looked for, but the stalk was certainly not strangulated.

Corner and Pinches (Case 3). F. C., male, aged forty years, was operated on for a huge, painful, and irreducible left inguinal hernia. It was found to consist of an enormous quantity of congested omentum, not strangulated, but adherent to the bottom of the sac. No torsion was noticed. But what then was the explanation of his having had painful attacks previously?

By means of these examples we hope to have shown that, from the knowledge previously gained, it has been possible to draw such inferences as are in favor or not in favor of torsion of the omentum being the key to the understanding of some clinical and pathological problems. Moreover, we hope that in such illustrations it has been possible to indicate the main directions in which our knowledge of the subject will be increased.

Differential Diagnosis. When the subject under consideration happens to be one the true clinical and pathological importance of which there is reason to believe is not yet understood, more stress must be laid upon its diagnosis or recognition at operation than upon its differential diagnosis beforehand. The concrete value of what we prefer to call the inferential diagnosis is to be placed far above the more abstract refinements of the differential diagnosis. Still, in view of the mistakes which have been made in the 53 cases collected in this paper we have thought it desirable that something should be said:

1. The instances of abdominal torsion, unaccompanied by the presence of any hernia, form about 10 to 12 per cent. of all cases of omental torsion. If the infrequency of the lesion is taken into account, as well as the fact that within the abdomen torsion is most unlikely to be overlooked, the subject of the differential diagnosis need not be considered in any detail. Five out of the six cases were diagnosed as appendicitis, with or without abscess. The sixth, occurring in Australasia, where obscure lesions are always guessed to be hydatids, was diagnosed accordingly. A tumor, or dulness with resistance, will always be present, and if found before the attack has commenced will raise other suspicions than those of

appendicitis. The condition is one which is to be recognized and treated properly during the course of operation.

2. When the torsion is in a hernial sac, 10 to 12 per cent., it will be found amongst the painful and irreducible, the incarcerated and strangulated herniæ. Here, as in the abdominal class, the surgeon should pay more attention to the recognition of the condition at operation than to its differentiation beforehand. A few words may be said upon the latter. In only two instances was bowel present, Mauclaire (47) and Makins (50). The first point to be proved will be that omentum alone is in the sac. In no case was the omentum strangulated; therefore it should have an impulse on coughing and, perhaps, bowel might be coughed down. The impulse given to an omentum hernia on coughing is far less definite than that communicated to one containing bowel. It is especially difficult to make satisfactory observations on large epiploceles which have an ample heaving impulse communicated to them by the muscles of the abdominal wall. In consequence, it is not easy to make sure of a large, non-strangulated, omental hernia. But if these two points are ascertained, then the accompanying symptoms are probably produced by torsion or epiploitis, of which two the latter is caused by the former. Beyond this the diagnosis is only a conjecture based on inference and such elementary differentiation as the above.

3. In the third division, which includes 75 to 80 per cent. of the cases, both the abdominal conditions and a hernial sac are present. The conjunction of these two enables us to take the differentiation farther than in the other classes; indeed, sufficiently far for a diagnosis rather than a conjecture to be made. Referring to what has been said above in considering the examples of abdominal and hernial torsion the signs of both will be found in the condition now under discussion. Typically, there will be present a tumor in the abdomen and an inguinal hernia, on the same side and of considerable duration (ten years), which will present signs of strangulation or irreducibility with pain. More than this, the tumor in the abdomen and the tumor in the hernia will be continuous. General symptoms of subacute peritonitis or intestinal obstruction will accompany these local signs. Such things as a local tuberculous peritonitis, an intraperitoneal abscess, pelvic cellulitis, or a subperitoneal lipoma might give rise to a somewhat similar state of affairs. But it is of interest to note that the majority of the diagnoses made concerned the hernial condition, *e. g.* strangulated epiplocele; and when the abdominal signs predominated, appendicitis was suspected. If the above signs and symptoms are found, and special attention given to the diagnoses which learned and competent observers have already made in examples of omental torsion, it should be possible to arrive with reasonable certainty at a diagnosis. There is some practical importance in this, because

it may be necessary to open the abdomen as well as the hernial sac. Demons' case (2) died, most probably because this was not done at first. Rectal examinations have never revealed anything in these cases and therefore will give very useful help in differentiation, *e. g.*, appendix abscess or pelvic cellulitis.

Strangulated inguinal herniæ are very infrequent in women; yet a relatively high percentage of cases of omental torsion, connected with a hernia, occurred in women. In middle-aged women one must always remember that the Fallopian tubes and ovaries, usually in an abnormal condition, are found in these herniæ. A rectal or vaginal examination enables one to make the distinction at once.

As omental torsion has never been found in a femoral hernia, the differentiation of it from a partial enterocele (Richter's hernia) need not be discussed.

In a certain number of cases the hernia has been reduced without relief of the symptoms, although the sac is empty. In these cases the obvious diagnosis of reduction without relief of the strangulation, or, as it is called, reduction *en masse*, is made, and nothing more can be inferred. Further, in a few more instances the attack or crisis came on while the hernial sac was empty, as in Peck's (7) and Noble's (45) cases. In these there is no guide to the fact that the case is not one of appendicitis; but as there had been a hernia of considerable duration present, the suspicion of omental torsion might arise.

The following list of the recorded cases up to the end of 1904 is arranged chronologically, and numbered so as to correspond with a brief record of each case published in the *St. Thomas' Hospital Reports of* 1904:

1. Max Oberst. Centralblatt f. Chir., 1882, pp. 441–447.
2. Demons. Rev. de chir., 1893, p 159.
3. Bayer. Centralblatt f. Chir., 1898, p. 462
4. Eiselberg. Deutsche med. Woch., 1898, p. 260.
5. Monod. Thèse de Requier, 1898.
6. Baracz. Deutsche Zeitsch. f. Chir., 1900, pp 584–589.
7. Peck. Medical Record, 1900. (Quoted by Wiener.)
8. Hochenegg. Wien. klin. Woch., 1900, pp. 291–293.
9. Wiener. Annals of Surgery, 1900, vol. ii p. 648–662.
10. Broca. Bull. Soc. chir , 1900, p 710; Chavanuaz, Gaz. hebd. méd. et chir., 1900, p. 601.
11. Heitz and Bender. Bull. Soc. anat , 1900, pp. 957–960.
12 Pothemt. Bull. Soc. chir., 1900, pp. 525–531.
13. Walther. Ibid.
14. Lejars. Ibid.
15. Lucas-Championnière. Ibid.
16. Wiesinger. Münch. med. Woch., 1901, p. 480.
17. Lucas-Championnière and Mauban. Bull. Soc. anat., 1901, p. 211.
18. Souliquot and Deschamps. Ibid., p 229.
19. Delivein. Wien. klin. Rundschau, 1901, p. 184.
20. Tuffier. Bull. Soc. chir., 1901, p. 547.
21. Wiart and Reuon. Bull. Soc. anat., 1901, p. 111.
22. Baldwin. Annals of Surgery, 1902, vol. ii. p. 940.
23. Syme Intercol. Medical Journal of Australasia, 1902, p. 444.
24. Riedel. Verhandl. der Deutsch. Gesel. f Chir., 1902, Bd. i. p. 92.

25. Moresco. Gaz. degli Osped. delle Clin., 1902, p. 693.
26. Heinlein. Münch. med. Woch., 1902, p. 1485.
27. Nordmann. Wiener klin. Woch., 1903, p. 459.
28. Nordmann. Ibid.
29. Quenu. Bull. Soc. chir., 1903, p. 520.
30. Rudolph. Wien. klin. Rundschau, 1903, p. 794.
31. Rudolph. Ibid., p. 811.
32. Rudolph. Ibid., p. 831.
33. Rudolph. Ibid., p. 848.
34. Tremolliere. Bull. Soc. anat., 1903, p. 693.
35. Blake. Annals of Surgery, 1903, pp. 99-102.
36. Sonnenberg. Arch. Internat. der Chir., 1903.
37. Zeller. Freie Vereinig. der Chir., Berlin, December, 1902.
38. Capette. Bull. Soc. anat., 1903, p. 539.
39. Rutherford Morrison. Northumberland and Durham Medical Journal, 1903, p. 211.
40. Vignard and Girandeau. Arch. prov. de chir., 1903, p. 206.
41. Malherbe. Ibid.
42. Barker. British Medical Journal, 1903, vol. i. p. 477.
43. Stewart. Journal of the American Medical Association, 1904, p. 767.
44. Audier. Thèse, Lyon, 1904.
45. Noble. American Journal of Obstetrics, 1904, p. 361; Medical and Surgical Monitor, Indianapolis, 1904, p. 261.
46. Mauclaire. Rev. de gyn. et de chir. abdom., 1904, p. 425.
47. Mauclaire. Ibid.
48. Scudder. Annals of Surgery, 1904, p. 916.
49. Trinkler. Deut. Zeitsch. f. Chir., 1904, p. 269.
50. Makins. St. Thomas' Hospital Reports, 1905.
51. Corner and Pinches. Ibid.
52. Corner and Pinches. Ibid.
53. Corner and Pinches. Ibid.
54. Michant. Trans. de la Soc. de chir., November 21, 1900.

CONGENITAL TRICUSPID STENOSIS COMPLICATED BY MITRAL STENOSIS.[1]

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The following instructive case recently came under my observation in my private course on physical diagnosis at the Metropolitan Hospital, New York City, and being one of rare occurrence I report the same in full:

S. S., admitted to the Metropolitan Hospital, New York City, September 13, 1904; Austrian, aged twenty-eight years; single; occupation, housework; height, 5 feet 6 inches; weight, 128 pounds.

Family history negative.

Birth natural. No instruments employed. Reared on the breast.

At six years of age had measles. She cannot recall any other illness in her life Upon this point she was most carefully interrogated. (Note questions under remarks at end of this article.)

[1] Read before the New York Pathological Society, January 11, 1905.

Moderate tea and coffee drinker; no drug habits. No history of venereal disease. Up to about six months ago has always enjoyed good health.

Recent Illness. About six months ago she began to be troubled with the following symptoms, although at this time there was no initial cause for the same. First noticed slight precordial pain with palpitation of the heart. Face and fingers began to get cold and bluish. At night had a slight, irritating, hacking cough, unaccompanied by pain. More or less light, clear, frothy mucous expectoration. At times slightly tinged with blood.

About six weeks ago began to notice slight swelling of lower extremities, which was much worse at end of day. Has noticed progressive weakness and increasing anxiety over her physical condition. Has noticed loss of weight and night-sweats regularly. Appetite is fairly good. Bowels move regularly. Urine is diminished in quantity.

Menstruation began at twelve years of age. Has always been regular and normal, never causing her any inconvenience until about four months ago, when she noticed that her periods ceased entirely without any apparent cause to her.

STATUS PRÆSENS. General build slight, but symmetrical. No noticeable abnormalities.

Nutrition. Panniculus adiposus greatly emaciated, musculature weak and flabby, skin and mucous membranes intensely cyanotic, and the eyelids, face, abdomen, and lower extremities show extensive œdema.

Skull. Well formed, giving no evidence of any abnormality.

Eyes. Left pupil slightly wider than right. Both react to both light and accommodation. Muscular action of eyeball normal. Visual field normal.

Buccal mucous membrane and pharynx of a dark-bluish color and covered by thick, tenacious mucus.

A visible diastolic presystolic pulsation of the jugulars. No evidence of any goitre or enlarged glands. Voice appeared husky. She frequently had to clear the larynx of a thick, tenacious mucus.

Chest. Form, long and fairly well rounded and symmetrical. Scapulæ slightly everted. Right shoulder lower than left.

Intercostal Spaces. Right apical (supraclavicular) well filled out. Left slightly depressed.

Expansion good with exception of right supraclavicular space. No evidence of any drawing in of intercostal spaces due to old pleuritic adhesions either upon inspiration or expiration.

Respiration. Costal in type.

Percussion. Right Lung: Absolute dulness at base line begins at fifth rib in the mammary line, fifth interspace in the mid-axillary line, and as seventh dorsal vertebra in the posterior

median line. General percussion note over the entire lung is hyperresonant. *Left Lung:* Slightly dull at apex. Dulness in the mammary line begins at the fifth interspace. In the midaxillary line at the sixth rib. In the posterior median line at seventh vertebra. General percussion note over the entire lung a clear hyperresonant tone.

Auscultation. Immediately above the line of absolute dulness posteriorly on both sides gave an amphoric respiratory sound. Numerous subcrepitant rales were heard over the base of both lungs. Expiration equalled in length the inspiration. Nowhere over either lung was any evidence of cavity formation or of tuberculous lesion. Examination for tubercle bacilli repeatedly proved negative.

Heart. Palpation. Apex beat at fifth interspace just inside the mammary line about two fingers in breadth. Plainly felt when patient leaned well forward, but not otherwise. Upon palpation at the apex could be detected a decided presystolic thrill. There was no epigastric pulsation.

Percussion. Absolute dulness extended to the right as far as the right sternal line and above as high as the lower border of the third rib close to the right sternal line. On the left side absolute dulness began at the upper margin of the fourth rib. Relative dulness reached 1 cm. to right of the right sternal line and on the left side, as high as the second intercostal space.

Auscultation. At the apex could plainly be distinguished a decided presystolic murmur, with a very loud accentuation of the second pulmonic sound. Auscultation over the mitral valve at the third interspace to the left of sternum gave a very loud, disagreeable presystolic murmur. Auscultation over the tricuspid valve was negative.

Pulse. Both radials were alike. The pulse was irregular, intermittent, soft, and compressible, averaging about 78 to the minute. None of the superficial arteries showed arteriosclerosis.

Abdomen. Palpation and percussion detected a small amount of ascites.

Liver. Lower margin of dulness reacts 5 cm. below the margin of the ribs in the mammary line. Delicate palpation over the edge of the liver gave a distinct impulse synchronous with the systole of the heart. Liver felt firm to the touch and its edges were well defined. Pressure upon the liver caused a heavy, dull pain.

Spleen. Slightly enlarged. At the margin of the ribs on left side in the midaxillary line its margin could be felt.

Urinalysis. Color, dark amber; reaction, neutral or slightly alkaline; specific gravity, 1026; quantity in twenty-four hours, 16 to 18 ounces; always under normal; albumin, slight trace by ferrocyanide test; sugar, negative; indican, negative; urea, 0.014 gm. per cubic centimetre; diazo reaction, negative.

Microscopic Examination. Frequently hyaline casts, bladder and vaginal epithelium; occasionally a few red blood corpuscles and leukocytes. Plenty of triple phosphates.

Diagnosis. Mitral stenosis with relative tricuspid insufficiency, accompanied by fatty degeneration of the heart muscle, producing the failing compensation with the secondary passive congestion of the internal organs. Hydrothorax, hydropericardium, and ascites and œdema of the lower extremities.

Subsequent History. (November 18, 1904.) Patient sits up in bed constantly. Apparently cannot lie down without great dyspnœa. Expectoration has become quite offensive. Constantly vomiting small quantities of blood of a coffee-ground appearance. Has attacks of hæmoptysis. Hands, legs, and eyelids extremely œdematous. Face, lips, and ears deeply cyanotic. November 25th, patient still expectorates blood and complains of much pain on the right side of chest low down as far as the fifth rib. Dyspnœa very marked.

Examination of middle and lower lobe of right lung showed marked dulness. From this date up to her death she continued to expectorate blood from time to time. Urine became more scanty, specific gravity reaching as high as 1031. Albumin in large quantity. Hyaline casts plentiful. Hemorrhagic infarct of middle and lower lobe of right lung was added to the diagnosis. Patient died December 9, 1904, at 12.30 A.M.

Autopsy performed December 10, 1904, at 12 noon. Post-mortem rigor in the extremities well developed. Hypostatic congestion in the dependent portions of the body. Face, lips, ears, eyes, fingers, and toes dark cyanotic hue. Pupils 4 mm. in diameter. Both the same. Lower extremities œdematous as far as middle of thigh. No evidences on the body.

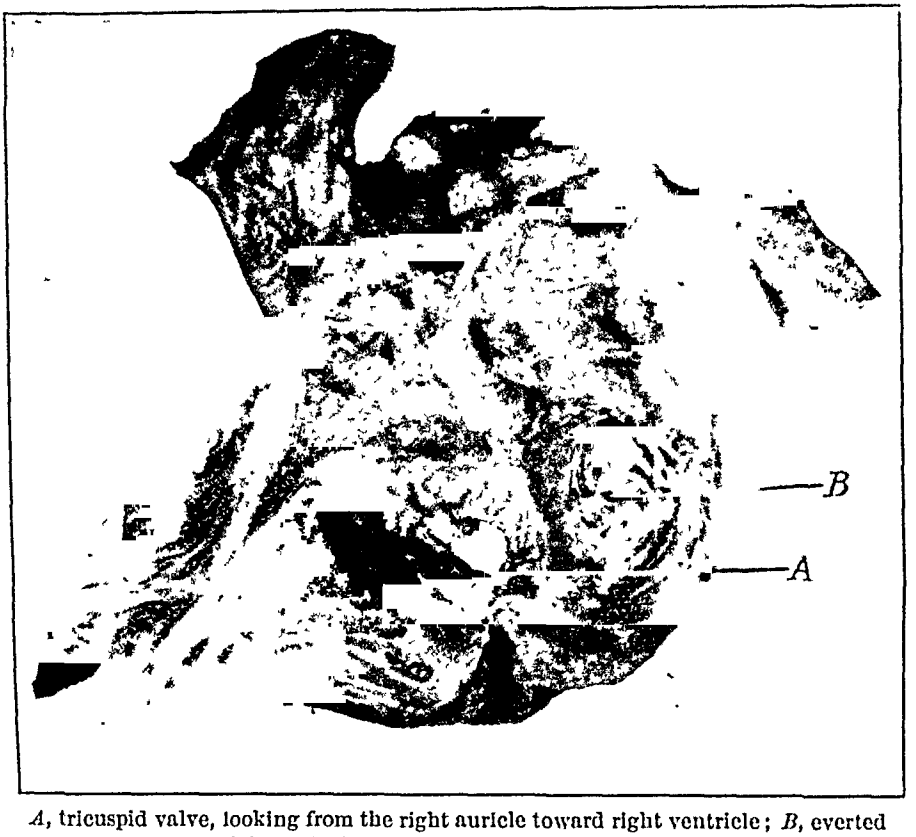
The abdominal cavity contained about two litres of a clear, dark-reddish transudate. Peritoneum was smooth, glistening, and transparent. Intestines appeared deeply congested. The vault of the diaphragm on the right side, at seventh interspace; on the left side, at the seventh rib. Right pleural cavity contained about three litres of a clear, dark-reddish transudate. Left pleural cavity contained about two litres of a clear, dark-reddish transudate. Slight adhesions between anterior border of left lung and the pericardium. At the apex of this lung were old pleuritic adhesions holding the apex firmly to the thorax walls. Pericardial sac contained about 500 c.c. of a clear, dark-reddish colored transudate. The two layers of the pericardium were everywhere smooth, glistening, and transparent.

Heart. Right auricle was extensively distended and filled with a dark, blue-black, non-coagulable blood.

Right ventricle appeared firm and but moderately distended. Left auricle was extensively distended and filled with a dark, blue-black, non-coagulable blood. Left ventricle was partially contracted and firm. The veins on the external surface of the heart

were intensely injected, the vena magna cordis at the base of the heart appearing as large as the first finger. The coronary arteries were not sclerotic The *heart muscle* was less firm than usual, and of a light-brown color. Papillary muscles streaked with yellow. Left ventricular wall averaged about 1 cm. Left auricular wall averaged about 3 mm. Right ventricular wall averaged about 4 mm. at the apex and measured 1¼ cm. at the attachment of the tricuspid valve. Right auricular wall averaged 2 to 3 mm. Circumference of aorta at aortic valve 6½ cm. Circumference of pulmonary at aortic

FIG. 1



A, tricuspid valve, looking from the right auricle toward right ventricle; *B*, everted right auricular appendix filled with thrombi.

pulmonary valve 7½ cm. Neither artery showed evidence of pathological change. Both right and left auricular appendices were greatly distended and filled with thrombotic masses.

A large, well-formed thrombus was found in the superior vena cava that was firmly adherent to the bloodvessel walls and was partly canalized. The external jugular vein was occluded by a thrombus.

The cusps of the tricuspid valve were united with each other (see Fig. 1), extending a distance from the base line toward their closing margins of 2½ cm. They were of normal thinness. There was no evidence of any present or previous inflammatory changes in or about this valve, so that the conclusion was forced that the condition present was *congenital.* The opening of the tricuspid

valve measured about 1½ cm. in diameter. The pulmonary valve showed no changes. The aortic valve showed a very slight union between the left and posterior cusps as well as slight fenestration of the posterior cusp. These changes were not of sufficient degree to have caused any impediment to the flow of blood. The mitral valve showed extreme stenosis of its two flaps so as scarcely to admit the tip of the finger. Along the base of the smallest anterior flap was a ridge of strong, hard, calcareous deposit (see Fig. 2)

FIG. 2



A, mitral valve; *B*, aortic valve; *C*, wall of ascending aorta; *D*, calcareous ridge along the anterior cusp of mitral valve.

extending for a distance of 3 cm. The closing margins of both flaps as well as the chordæ tendineæ greatly thickened. The balance of the endocardium everywhere appeared smooth, glistening, and transparent.

Right Lung. External surface smooth. Pleura covering same was clear and transparent. Lung appeared voluminous. Middle and lower lobes were firm to touch, and of a dark-blue color. Along the anterior margin of the upper lobe was a firm, circumscribed, dark-blue prominence, measuring fully 3 cm. in diameter,

which upon cut surface proved to be a recent hemorrhagic infarct. Upon the cut surface it was found that the entire middle lobe and the lower four-fifths of the lower lobe (see Fig. 3) were two large, recently formed hemorrhagic infarcts. The bloodvessels that supplied these areas were filled with firmly adherent thrombi (*autochthonous thrombosis*). The glands at the hilus of the lung appeared normal. Connective tissue not increased.

Left Lung. At the apex were old pleuritic adhesions firmly uniting the apex to the thoracic walls. The balance of the pleural covering appeared normal. The entire apex of this lung proved to be involved

FIG. 3



A, bloodvessels of lower lobe of right lung filled with autochthonous thrombi) the lobe of lung is bisected); *B*, remains of lower lobe not affected by the hemorrhagic infarct; *C*, part included by hemorrhagic infarct in lower lobe; *D*, middle and upper lobe drawn back.

in an old hemorrhagic infarct that was now undergoing putrid necrosis. The balance of this lung upon cut surface showed passive congestion and acute œdema.

Spleen. Weight 8 ounces; firm in consistency; dark mahogany brown color; capsule slightly thickened; trabeculæ increased. Pulp firm; bloodvessels congested (cyanotic induration).

Liver. Displaced downward 5 cm. below costal margin of the ribs. Weight 5 pounds. Color mottled yellow and brown; a decided nutmeg appearance. Firm in consistency. Slightly granular. Bloodvessels congested. Lobules plainly visible, dark brown in centre and surrounded by a yellowish boundary (cyanotic indurative fat liver).

Kidneys. Left: Weight 8 ounces. Dark-brown color; firm in consistency; capsule readily stripped. Externally smooth. Blood-vessels of pyramids deeply congested. Cortex measures 6 to 10 mm. Markings of cortex plainly visible. Also the Malpighian corpuscles. Cyanotic passive congestion.

Right: About one-third larger than normal. Externally variegated in color from deep chrome yellow to dark brown. Firm in consistency. Innumerable *anæmic and hemorrhagic infarcts.* Upon cut surface the larger arteries were filled with firmly adherent thrombi (autochthonous). Microscopic sections of this kidney showed many anæmic and hemorrhagic infarcts, with the end arteries filled with well-formed and organized thrombi, in some cases showing canalization. Query: Why this condition in the one kidney and none similar in the other?

Adrenals showed no change.

Uterus about size of uterus in the third month of pregnancy. Walls were firm and hard. Mucous membrane heavily congested. Chronic metritis.

Stomach and Intestines. Walls were deeply congested and covered with a thick, tenacious mucus. Chronic gastritis and enteritis.

Diagnosis. Tricuspid stenosis complicated by mitral stenosis, together with fatty degeneration of the heart muscle. Hemorrhagic infarcts of both lungs, together with passive congestion and œdema. Anæmic and hemorrhagic infarcts of the right kidney. Passive congestion and cyanotic induration of the internal organs, especially liver, spleen, and left kidney. Hydrothorax, hydropericardium, and ascites.

REMARKS. It is interesting to note the diagnosis made of relative *tricuspid insufficiency* as based upon the mitral stenosis, greatly accentuated second pulmonic sound (enlarged right heart), venous and liver pulse; whereas to the contrary we are dealing with a *tricuspid stenosis,* which I am frank to admit did not enter into my calculations at the bedside.

Reviewing the history of this case, and taking into consideration the severity of the mitral lesion, it appears difficult to harmonize the two. Did the early attack of measles (six years of age) furnish the etiological factor wanted? Can such lesions arise *sui generis* or from no cause that a patient with average intelligence would likely perceive? Did the tricuspid stenosis (congenital) have any etiological bearing upon this extensive mitral lesion?

Is it not curious that a congenital stenosis of the tricuspid valve as severe as this one (opening 1½ cm. in diameter) should have caused no noticeable disturbances throughout twenty-eight years of the patient's life, until the mitral valve had become so seriously crippled?

The writer wishes to acknowledge his indebtedness to Mr. Chas. Brush, the hospital photographer, for the photographs he has furnished me in this case.

REVIEWS.

ÆQUANIMITAS; WITH OTHER ADDRESSES TO MEDICAL STUDENTS, NURSES AND PRACTITIONERS OF MEDICINE. By WILLIAM OSLER, M.D., F.R.S. Philadelphia: P. Blakiston's Son & Co., 1904.

THIS book may be regarded as the parting gift of its distinguished author to his professional brethren in America, and no valedictory could be more graceful or of more genuine value. The addresses contained in it cover a wide range, having been delivered on very different occasions, and before audiences of distinct types. Whether the address is to a body of students about to graduate, or nurses just entering upon their professional duties; to a group of students of medical history, or to a distinguished scientific body, it may always be counted upon to contain many of those precious pearls of wisdom which the greatest peripatetic teacher of his day always dispenses with such a generous hand. Most of them are, or ought to be, familiar to medical readers through the columns of the medical journals in which they have heretofore appeared, but this fact we are sure will make their appearance in book form all the more gladly welcomed. The longest article, that on "Medicine in the Nineteenth Century," appeared in the New York *Sun*, and was republished in book form in a symposium of articles on the progress made in various lines during the nineteenth century, apropos of the advent of the twentieth. It has accordingly reached somewhat the lay public for whom it was intended. It contains a most excellent summary of the progress made in medicine during its greatest centennial, but especial praise should be accorded to it because of the characteristic manner in which its author has presented to the layman the advantage he derives from all the wonderful achievements of medical science, and indicates their practical value to the world at large. In other words, Dr. Osler emphasizes not only the humanitarian but also the commercial value of the labors of medical science.

Of more academic interest is the paper on "Physic and Physicians in Plato," depicting the Greek physician as drawn by his contemporaries. Dr. Osler loves to hark back to whatever was best in the olden days, and this essay shows a love of its subject which beautifies and lends an interest to what to many of we moderns

would otherwise be as dry leaves. To every man in the profession the address on the "Masterword in Medicine" will have an infinite meaning. The masterword which opens the gate to success is "work." To him who hath it shall be given the due reward, but he who fails to achieve the masterword shall inevitably be cast into outer darkness. Another address is in the nature of a solemn warning against professional narrowness which unfortunately mars the breadth of view of so many otherwise excellent men. It is that on "Chauvinism in Medicine." It warns against narrowness, whether "national, provincial, collegiate, or personal." Among its great merits are the fearlessness with which it directs attention to several points in which professional narrowness manifests itself and the excellence and simplicity of the remedies for these conditions which it suggests.

In the several addresses to students contained in this volume there will be found a storehouse of wisdom of which it is sincerely to be hoped all generations of future doctors will avail themselves. The address from which the volume takes its title is an able disquisition on the advantage to the medical man of possessing imperturbability of mind. He urges the young man at the outset of his career to cultivate this faculty with assiduity, as its practice will be of the greatest value not only to him but to his patients. In another address to a student body entitled "Teacher and Student," he tells the young men to acquire first the Art of Detachment, second the Virtue of Method, third the Quality of Thoroughness, and lastly the Grace of Humanity. Dr. Osler's advice is couched in such graceful language and is of such evident wisdom that it is sure that many a reader as well as hearer must have felt a call to higher things as it reached his heart.

There are two addresses delivered to graduating classes of trained nurses, one called "Doctor and Nurse," the other "Patient and Nurse." The latter is the longer and will be read with interest not only by nurses and doctors, but also by any of the laity who may have been brought more or less in contact with the trained nurse.

There are several addresses delivered to the Association of Medical Librarians, and at the opening of medical libraries, which suffice to show Dr. Osler's great love for and acquaintance with books. At the end of the book is appended a list of ten books constituting a "Bedside Library for Medical Students." Let us hope that the words of so charming a guide will prompt at least a large proportion of students to adopt his suggestions for their literary pabulum. To this list we would like to add "Æquanimitas," by William Osler, a book which the sooner they become familiar with from cover to cover the better it will be for them.

F. R. P.

THE PRINCIPLES OF HYGIENE: A PRACTICAL MANUAL FOR STUDENTS, PHYSICIANS AND HEALTH OFFICERS. By D. H. BERGEY, A.M., M.D., Assistant Professor of Bacteriology, University of Pennsylvania. Illustrated. Second edition, thoroughly revised and enlarged. Pp. 536. Philadelphia, New York, and London: W. B. Saunders & Co., 1904.

THE first edition of this valuble hand-book was exhausted in a little more than two years, and during that time the progress of sanitary science was so great that a not inconsiderable part of the text became either wholly obsolete or more or less behind the times. The present edition shows extensive pruning and many additions of new material, and it presents the subject in the very latest light and in a most attractive way. All that was said in praise of the first edition can be repeated with hearty emphasis, and especially is this true of the chapter on Vital Causes of Diseases, which embraces consideration of the all-absorbing topic of immunity, of the relation of insects to human diseases, and of animal and vegetable parasites; but it must be said that a fuller discussion of the parasitic diseases which recent investigation has shown to play such important parts in the South and in many foreign countries might well have been given some of the space accorded to the diminishingly important subject of Quarantine, which takes up one-seventh of the entire book. The index, although sufficiently voluminous, is hardly as helpful as it should be: it includes very many items which one would never think of looking for, and gives far too little space to most of the really important subjects. C. H.

SAUNDERS' QUESTION COMPENDS, No. 3. ESSENTIALS OF ANATOMY, INCLUDING THE ANATOMY OF THE VISCERA ARRANGED IN THE FORM OF QUESTIONS AND ANSWERS PREPARED ESPECIALLY FOR STUDENTS OF MEDICINE. By CHARLES B. NANCREDE, M.D., Professor of Surgery and of Clinical Surgery in the University of Michigan; Emeritus Professor of General and Orthopedic Surgery, Philadelphia Polyclinic, etc. Seventh edition, thoroughly revised. Philadelphia, New York, and London: W. B. Saunders & Co., 1904.

THE mere fact that this is the seventh edition of this book would indicate its popularity. It is one of the best of all the question compends and will no doubt continue to enjoy its deserved success. Considering the size of the book the illustrations are most satisfactory. Excepting for the rewriting of the chapters on the Nervous System and the correcting of slight former errors the book remains the same as in its previous editions. J. H. G.

ANATOMY APPLIED TO MEDICINE AND SURGERY. By D. E. MUNDELL, B.A., M.D., Professor of Applied Anatomy, Faculty of Medicine, Ontario University; ex-Examiner Practice of Medicine, Ontario Medical Council; Surgeon to Kingston General Hospital. Kingston: Printed at the *British Whig*, 1903.

THIS book is especially prepared for the use of students, being a means of teaching them to practically apply their knowledge of anatomy. It would appear to be a most valuable aid. The various regions of the body are taken up separately, the anatomy being dealt with briefly and the injuries and surgical diseases of the part outlined, especially the surgical operations. The illustrations are not very numerous nor are they particularly good, but of course the student would employ this book only as an auxiliary to his larger works on anatomy, and hence illustrations are not so necessary. The text is very comprehensive, and we believe that, on the whole, not only the student but the occasional operator will find this an invaluable little volume. It is to be regretted that the list of *errata* is so extensive. J. H. G.

THE OPHTHALMIC YEAR BOOK. A Digest of the Literature of Ophthalmology, with Index of Publications for the Year 1903. By EDWARD JACKSON, A.M., M.D., Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic; President of the American Academy of Ophthalmology and Otolaryngology; Ophthalmologist to the Denver County Hospital, St. Anthony's Hospital, and Mercy Hospital, Denver, etc. With 45 illustrations. Denver, Colorado: The Herrick Book and Stationery Co., 1904.

THIS is a most valuable contribution to current ophthalmology. The oculist who desires to be informed in regard to the work which is being done and the progress made in his specialty (and who does not?) is compelled to devote an immense amount of time to the study of periodical literature; and it is not everyone who can have access to the necessary material. One need only call to mind the voluminous Jahresbericht of Nagel, and even this is incomplete, to have an idea of the mass of printed matter annually sent forth relating to ophthalmology. It is out of the question for the average practitioner to attempt anything like an adequate review of this mass of literature. It requires really an expert to sift out the useful. This is exactly what Dr. Jackson has admirably accomplished. He has first of all selected the most important contributions which have appeared during the year, not as extracts, but sufficiently in detail to make them readily intelligible, a result which has been

greatly aided by the illustrations which the book contains. A particular value is given to the matter by the criticisms and explanations which the author interpolates on almost every page. Papers which find a place in this digest are not chosen simply because of their novelty, but for their utility in furthering the writer's object.

The book also contains a list of books and monographs and the more important journal articles which have appeared during the year.

The work divides the subject of ophthalmology as follows: refraction, ocular movements; then follow diseases of the different ocular structures, beginning with the conjunctiva, with special chapters upon the pupil, glaucoma, tumors, injuries to the eyeball, and general ophthalmology and operative measures. A very useful index completes this admirable book.

We would strongly urge the merits of this work upon every practitioner of ophthalmology. It is particularly useful to one who intends to make written contributions to the subject. Such a one can here find in a few moments information which would consume much time to discover elsewhere. T. B. S.

THE OPEN-AIR TREATMENT OF PULMONARY TUBERCULOSIS. By F. W. BURTON-FANNING, M.D. Cantab., Physician to the Norfolk and Norwich Hospital; Honorary Visiting Physician to the Kelling Open-air Sanatorium. London, Paris, New York, and Melbourne: Cassell & Co., Limited. Chicago: W. T. Keener & Co., 1905.

THE author of this most excellent treatise on the subject of the sanatorium and open-air treatment of tuberculosis gives an exhaustive detailed account of the method in use at the Kelling Sanatorium, with which he is connected, comparing it also with that in vogue in other countries. He points out the possibility of conducting proper treatment in the home as well as in the sanatorium, even in a crowded city, with a fair hope of success.

Altitude and climate appear to have much less to do with a cure than was formerly supposed, and except in special indications any climate, cold or warm, dry or damp, may suffice for the treatment provided constant fresh air, the *sine qua non*, be supplied both night and day. Much of the success of the treatment depends upon the thorough understanding of the relations of rest, diet, exercise, and open-air life upon the pulse, temperature, and bodily weight of the patient. The results are classified by Dr. Burton-Fanning under five headings: I. Recovery with fitness for work. II. Arrest of disease with fitness for a certain degree of work. III. Arrest, but with the necessity of continuing an invalid life. IV. Improvement. V. No improvement.

A cure can only be pronounced after a man is dead, as Dr. Chidell says. Finally a point of great importance in the system is dealt with from a very practical aspect, and that is, the question of the after-life and employment of the recovered patient.

The sanatorium treatment must only be regarded as a preliminary to a most carefully regulated after-life, so that in order that the results may be lasting a certain amount of consideration and latitude from the employers must be asked for those patients who return to work.

Too great stress cannot be laid on the value of an early diagnosis in its relation to result. The author is to be congratulated on his excellent presentation of so important a subject. J. N. H.

LES DEGENERESCENCES DES FIBROMYOMES DE L'UTERUS. By G. PIQUAND, *Ancien Interne Laureat des Hôpitaux, Prosectem* provisoire à la Faculté de Médecine. 426 pages. Paris: G. Steinheil, 1905.

IN this interesting monograph are included a number of interesting facts and observations which have hitherto been scattered throughout the literature. While it is based on only 42 cases, observed in Quenn's clinic, the author has shown a wonderful amount of patience and industry in reviewing the literature of the subject and excellent judgment in generalizing from the facts collected.

The book is divided into two parts; the first, which deals with non-infectious degeneration, including seven chapters on fibrous degeneration, calcareous, œdematous, sarcomatous, telangiectatic, epithelial, and cystic. Under part second are included two chapters on suppuration and gangrene, respectively. The same order is followed in the arrangements of the paragraphs in each chapter—viz., history, pathological anatomy, pathogeny, clinical symptoms, diagnosis, and prognosis. The references are exhaustive, and must be of great value to any subsequent writer on this subject.

Space does not allow us to enter into a detailed review of this monograph, but we can only note some of its most interesting points. For example, the chapter on the origin of epithelial elements in fibromyomata. The author concludes, from his personal observations, that they are derived from the endometrium, but he admits they may exceptionally result from proliferation of the remains of the Wolffian and Müllerian ducts. He states, as a fact not to be disputed, that malignant degeneration of these glandular elements in the midst of the fibromyomatous tissue does occur. In an interesting section on the coexistence of fibromyoma and cancer of the uterus no less than 112 cases are cited.

The final conclusions arrived at by the writer are as follows: In 30 out of 100 fibroid tumors of the uterus, some form of degeneration has been noted, the prognosis varying widely according to the changes which are present. Fibromatous degeneration is the most common and least serious; sarcomatous the most grave. The climacteric period is the favorite time for such changes in the tumor. This is directly opposed to the popular view that the menopause is favorable to retrograde changes and atrophy of these neoplasms, and supports the opinion of those who contend that they should be removed before this period. H. C. C.

MALFORMATIONS OF THE GENITAL ORGANS OF WOMEN. By CHARLES DEBIERRE, Professor of Anatomy in the Medical Faculty at Lille. With 85 illustrations. Translated by J. HENRY C. SIMES, M.D., Emeritus Professor of Genitourinary and Venereal Diseases in the Philadelphia Polyclinic. Pp. 182. Philadelphia: P. Blakiston's Son & Co., 1905.

THE translator states in his preface that his object has been "to fill a void in English medical literature." Whether the reader will agree with him or not is doubtful. Chapter I. deals briefly with the normal anatomy of the genital organs and breasts and presents no new or interesting facts, a criticism applicable also to the brief chapter on development. In Chapter III., on malformation, the ovaries are first considered, then the tubes, round and broad ligaments, the uterus, vagina, external genitals, and breast.

Malformation of the vagina (and especially of the hymen) seems to be the author's favorite topic, as he devotes considerable space to it, apparently because it affords him the opportunity to introduce a number of prurient details and questionable anecdotes, which are related in true Gallic style, though they must be offensive to English readers. Hermaphrodism is treated most superficially; also anomalies of the breast.

The final paragraph of this amusing monograph is a sufficient commentary on its supposed *raison d'être:* "Our general conclusion from the study of the anomalies of the genital organs of woman is that in nature nothing is unusual." H. C. C.

TRANSACTIONS OF THE TWENTY-EIGHTH ANNUAL MEETING OF THE AMERICAN DERMATOLOGICAL ASSOCIATION, 1904. By C. J. WHITE, Secretary.

THE proceedings of this and other similar national associations devoted to the consideration of special branches of medicine and surgery are becoming more valuable with each year, and the hand-

some volume of over 200 pages before us goes to confirm this statement. It contains many excellent articles, good photographs of cases, and photomicrographs. Of the latter, special mention may be made of the beautiful work in this direction of Dr. J. A. Fordyce, of New York, illustrating his paper on "Affections of the Mucous Membrane in their Relation to Skin Diseases." Articles of this kind are of general interest and serve to bring this specialty, now so firmly planted, in close relation to general pathology. M. F. Engman, of St. Louis, also has a paper of merit on the same subject. W. T. Corlett, of Cleveland, discusses "Postvaccinal Eruptions," and Hyde and McEwen, of Chicago, the "Relation of Certain Dermatoses to Changes in Vascular Equilibrium." Dr. W. F. Breaky, of Ann Arbor, calls attention to "Parasitic Sycosis Communicated from Cattle," a subject which we think is not so well recognized by the profession as it deserves. There is no doubt that many contagious diseases of this kind are contracted from animals, especially dogs, cats, and horses. Perhaps the most valuable contribution to the *Transactions* are the elaborate tables showing the combined returns of cases of cutaneous disease for the preceding year, comprising no less than 25,000 cases observed and reported by the members of the Association. The volume is admirably edited by the Secretary, Dr. C. J. White, of Boston, and reflects credit upon all those who took part in the meeting. It should have a place in all medical libraries as a work of reference. L. A. D.

LE CORYZA SYPHILITIQUE, RHINOPHARYNGITE PURULENTE SEPTICO-PYOHENIQUE, CHEZ LES NOUVEAUNES SYPHILITIQUES. Par M. LE DR. PAUL GASTOU, Ancien Chef de Clinique et Assistant de Consultation, Chef de Laboratoire de la Faculté à l'Hôpital Saint-Louis. Paris: O. Doin, éditeur, 1904.

DR. GASTOU here presents in pamphlet form, reprinted from the *Revue d'hygiène et de médecine infantiles*, 1904, tome iii., No. 3, a very complete study of one of the principal manifestations of hereditary syphilis in early infancy. The text is illustrated by four full-page plates, two of which are in color, showing the facies of an infant suffering from a severe grade of the disease, and various microscopic sections of the healthy and diseased mucous membrane of the nose, and sections of lung affected with pneumonia secondary to the nasal disease. It forms a complete monograph on a subject that in the text-books is usually dismissed with a few lines of vague description, and thus constitutes a valuable contribution to our knowledge of the disease. T. S. W.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D.,

REGIUS PROFESSOR OF MEDICINE, OXFORD, ENGLAND,

AND

W. S. THAYER, M.D.,

ASSOCIATE PROFESSOR OF MEDICINE, JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND.

Symptomatology and Diagnosis of Vincent's Angina.—VINCENT, who first described this throat affection in 1898, in a recent article (*Lancet*, May 18, 1905, p. 1260) gives a concise account of its chief features. He describes it first under the name of "d'angine à spirilles et bacilles fusiformes." It is comparatively common and occurs most frequently in children between six and ten and in adults between eighteen and thirty years of age. From statistics it constitutes 2.26 per cent. of all forms of throat infections. Those living in bad hygienic surroundings, those with generally impaired health, and those with carious teeth and who take poor care of their mouth are most liable to contract it.

Two organisms are found in the smears and cultures from the throat. One is a long fusiform bacillus. The average length is from 6 to 8μ, the longest being 10 to 12μ. The breadth varies from 1 to 1.5μ. It may or may not be motile. The protoplasm is frequently vacuolated. The bacillus does not stain by Gram's method. The other organism is a very long, delicate spirillum, which is actively motile in fresh specimens. This organism usually but not always accompanies the bacillus. While these two organisms are regarded as the cause of this form of sore throat, other organisms, such as the staphylococcus, streptococcus, or bacillus coli, may be also present.

Clinically, the disease manifests itself in two forms: One, the diphtheroid form, is rare and constitutes only 2 per cent. of the cases and closely simulates diphtheria; the other, and by far the most common is the ulceromembranous form. A grayish or yellowish membrane appears on one of the tonsils and about the fourth day ulceration begins and a deep excavation of the tonsil occurs. The ulceration is bilateral in about 2 per cent. of the cases. The throat is sore and deglutition difficult. There is fever, general pains, and an enlargement of the glands of the neck.

The disease has to be distinguished from diphtheria and syphilitic ulceration of the tonsils. This is readily done by the bacteriological examination of the throat exudate. The treatment consists in painting the throat twice daily with tincture of iodine, which is almost a specific, and clears up the local inflammation in a few days.

Experimental Measles.—HEKTOEN (*Journal of Infectious Diseases*, March 1, 1905, p. 238) reviews the literature on the experimental production of measles in man from the first recorded attempt by Francis Home, of Edinburgh, in 1758, to the present date, and concludes that where positive results were obtained sufficient care was not taken to exclude the possibility of the disease having been contracted in the usual way.

With the consent of two subjects he sought to transmit the disease experimentally, and in both instances met with positive results. The procedure was in brief as follows: Blood was obtained from one of the elbow veins of two individuals suffering from measles in the acute stages. Ascites-broth was inoculated with some of this blood and allowed to remain in a thermostat for twenty four hours at a temperature of 37° C. From 4 to 5 c.c. of this inoculated ascites-broth were then injected subcutaneously into two healthy persons, who were in such surroundings that accidental infection was considered impo sible. In the first case the blood was obtained from a patient on the fourth day after the appearance of the rash. The subject inoculated, a medical student, developed fever on the th'r eenth day, and suffered from an uncomplicated attack of typical measles. In the second case the blood was obtained from a patient thirty hours after the earliest appearance of the rash. The inoculated subject commenced to have fever on the eleventh day, and on the fourteenth day a typical measles rash appeared.

The blood cultures from both of the original cases remained sterile so far as bacteria demonstrable by the usua methods were concerned.

Hektoen states that the experiments permit the conclusion that he virus of measles is present in the blood of patients with typical measles, sometimes at least during the first thirty hours of the eruption; further, that the virus retains its virulence for at least twenty-four hours when such blood is inoculated into ascites-broth and kept at 37° C. He asserts that the demonstrat on shows that it is not difficult to obtain the virus of measles unmixed with other m'crobes and in such form that it can be studied by various methods.

A Case of "Splenomegalic" or "Myelopathic" Polycythæmia with True Plethora and Arterial Hypertonia without Cyanosis.—F. PARKES WEBER (*Lancet*, May 13, 1905, p. 1254) reports such a case occurring in a Jewish woman, aged thirty-seven years, who first came under observation in July, 1903, for acute erythromelalgia of the left foot. The case differed from those of splenomegaly accompanied by polycythæmia reported by Vaquez, Saundby and Russell, Rosengart, Osler and others, in that cyanosis was not present. The cutaneous blood-vessels were somewhat overfilled, however. The splenic enlargement was not marked, the border being felt one to two fingers' breadth from the costal margin. The red cells ranged between 8,016,000 per c.mm., the lowest to 10,960,000 per c.mm., the highest count. Normoblasts were present. There was usually a leukopenia. The relative percentage of polymorphonuclears was very high, ranging between 66 and 81 per

cent. The hæmoglobin was between 120 and 184 per cent. The blood pressure and total volume of red cells were both increased. There was also a striking increase in the specific gravity and viscosity of the blood, With the exception of improvement in the associated erythromelalgia treatment had practically no effect on the patient's condition.

From a study of a case previously reported by the writer, in which an autopsy was obtained, the opinion is expressed that the disease is always accompanied by and at all events mainly due to an excessive pathological activity in the bone-marrow. This was evidenced by the extensive conversion of yellow into red bone-marrow. Weber thinks the high percentage of polymorphonuclears in the present case is confirmatory of this view.

The sequence of events in these cases and their causal connection is believed by the writer to be as follows: 1. Increased erythroblastic activity involving a great part but not necessarily the whole of the bone-marrow. 2. Increased viscosity of the blood resulting from the polycythæmia. 3. Dilatation of small bloodvessels, partly to lessen resistance to the abnormally viscous blood, partly to make more room for dilution of the blood. 4. The "plethora vera," or "polyhæmia," is probably to be regarded as an attempt to compensate for the increased viscosity of the blood and for the excessive percentage of the total blood volume occupied by the cells. 5. The arterial hypertonia is to be regarded as a result of the greater strain thrown on the circulatory system. 6. Cyanosis, when this occurs, is probably due to inadequacy of the series of compensatory changes, which, according to his view, precedes it.

The enlargement of the spleen is held to be due to the plethora vera rather than to excessive functional activity in attempting to compensate for the excessive production of red corpuscles by increased destruction. Weber thinks that the association between the polycythæmia and the erythromelalgia may have been merely an accidental one, although he calls attention to the fact that a similar association has been noted in cases reported by Türck and W. Weintraud.

SURGERY.

UNDER THE CHARGE OF

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Cases Illustrating Alternative Operations for Intestinal Obstruction.—BARKER (*British Medical Journal*, April 8, 1905, p. 756) in the first case opened the distended bowel above the obstruction (growth of the cæcum), allowing the contents to escape, after which an ileocolostomy was done, short-circuiting the obstruction. At the end of six weeks,

when everything was quiet, the growth with the cæcum, ascending colon, and about six inches of the ileum were successfully excised.

In the second case, cancer of the splenic flexure of the colon, because of the acrid feces and toxins in the gut and the sodden and rotten condition of the gut, which would hardly hold stitches, it was considered inadvisable to short-circuit, and an artificial anus was made. Ulceration of the abdominal wall from the feces followed, and rendered the anastomosis of the bowel above the growth to the sigmoid, which was done later, a dangerous operation. Still later the splenic flexure with the growth was excised, and finally, at another operation, the artificial anus was closed by incising around it, closing the proximal end and dropping it back into the abdomen, the distal end being sutured in the lower angle of the abdominal incision. This permitted the escape of gas and mucus from the cæcum and colon, which had been closed at the other end. The patient improved and left the hospital very well.

In the third case, a diabetic, a cancer of the transverse colon, in which the obstruction had never been acute, was excised and the divided bowel joined "end to end." There was no need of a preliminary artificial anus. Recovery, in spite of the 6.6 per cent. of sugar, followed.

In the fifth case short-circuiting was done around an apparently malignant growth of the small intestine, but although six years have passed since, the patient remains perfectly well and shows no signs of the presence of the growth. All those who saw it thought it to be a sarcoma. In another case short-circuiting was done through a second incision in the abdominal wall, permitting the removal of an incarcerated and blackened femoral hernia and the immediate performance of a radical cure.

A Case Simulating Intracranial Tumor in which Recovery Was Associated with Persistent Cerebrospinal Rhinorrhœa.—T. R. GLYNN and E. E. GLYNN (*British Medical Journal*, April 22, 1905, p. 871) report the case of a young man, aged twenty-one years, who struck his head violently against a beam. Intracranial symptoms began then and continued to grow worse, until about five years later clear fluid began to escape in drops from his right nostril. Immediately he commenced to improve, his mental vigor and muscular strength returned, and his sight, which had been lost, was restored. The progress toward recovery was rapid.

Examination of the nasal cavity showed it to be normal and the escaping fluid to be cerebrospinal,

That spontaneous discharge of this fluid can occur through the nose is not hinted at in the text-books. The cause of the symptoms was probably an internal hydrocephalus. The blow on the head, the writers believed, excited a slight partial meningitis in the region of the fourth ventricle, leading to occlusion of the foramen of Majendie and retention of the fluid within the ventricles. The fluid probably forced its way through the thin roof of the fourth ventricle and along the sheath of the olfactory nerve into the lymphatic network of the mucous membrane of the nose and so to the epithelial surface.

Subacute Perforation of the Stomach, with Report of Three Cases.—LUND (*Boston Medical and Surgical Journal*, May 4, 1905, p. 516) says that the size of the perforation will be in proportion to the severity

of the onset of the symptoms. In the so-called subacute perforations the very small opening may become plugged by a bit of omentum or fibrin. Under proper treatment the fibrinous adhesions may become organized into fibrous tissue, the ulcer heal, and no symptoms be left, except those due to the presence of the adhesions. On the other hand, the filmy adhesions may be digested away, the peritonitis may spread, and at the end of two or three days develop rapidly, and perhaps end the patient's life in a few hours.

The writer's cases belong to the first class. Owing to the difficulty in diagnosis the only method of settling the question is by an exploratory laparotomy. The diagnosis may be confused with acute cholecystitis or ulcer of the colon, but in all three conditions, when perforation is threatened, operative treatment is the only one to be considered.

Conclusions: 1. The symptoms of subacute perforation are similar to those of acute perforation, with the important exception that they are less violent and are not followed by collapse or by the development of general peritonitis.

2. The location of the pain and tenderness depend upon the location of the ulcer and varies with it.

3. The treatment should be, if possible, posterior gastroenterostomy, without breaking up the protective adhesions.

Some Suggestions in Regard to the Diagnosis of Seminal Vesiculitis.—CABOT (*British Medical Journal*, May 11, 1905, p. 542) thinks that the diagnosis of seminal vesiculitis is frequently made on insufficient grounds. The presence of infiltration and inflammatory exudation should be shown, if by the termination "itis" true inflammation is meant.

The symptoms considered are distention and thickening of the vesicle, with tenderness of the vesicle and perivascular tissues. The observations of Simons and Duhat show that a certain amount of distention of the vesicle is physiological. Not infrequently it may show more than ordinary distention from chronic prostatitis, in which the seminal vesicle is not involved.

Thickening or stiffening of the vesicle is difficult to determine. No report should be considered conclusive unless made on the results of more than one examination or by one very expert in rectal examination, and it should be insisted that a marked degree of thickening should be regarded as conclusive evidence.

In estimating the tenderness it is difficult to establish the normal sensitiveness in this region, which varies immensely in different individuals. Therefore, while valuable in some cases as contributory evidence, it can hardly be regarded as an important symptom except in acute cases.

Cabot considers examination of the contents of the vesicle as the most reliable evidence. From a series of 19 cases thus examined, he found in only 3 cases pus in more than negligible quantities, and in these both inflammatory thickening and tenderness were present. The absence of pus in the other 16 cases shows that either inflammation of the vesicle was not present or it was not characterized by pus formation.

Action of the X-rays upon the Blood.—MM. AUBERTIN and BEAUJARD have, in their studies upon the action of the *x*-rays upon the

hæmatopoietic system, found that an animal subjected to the rays exhibits a polynuclear leukocytosis due to an excessive production of leukocytes. Applied over a small area the rays produce a transient leukocytosis. Repeated applications over the same area cause an excitation of leukocytosis, more and more marked. The red cells become diminished and altered, and, finally, nucleated red corpuscles appear, as well as myelocytes. Then the polynuclear leukocytes diminish, leaving a "leukohæmia," with myelæmia. The red marrow subjected to the rays becomes more fatty than normal, the remaining myeloid tissue is in a state of hyperactivity, and the spleen is the seat of a myeloid change. In conclusion, the observers state that in the healthy animal, as in the leukæmic patient, emigration and destruction of leukocytes preceded by an increased leukocytosis and final degeneration of the tissue exposed to the rays take place.—*Le progrès médicale*, 1905, No. 6, p. 86.

THERAPEUTICS.

UNDER THE CHARGE OF

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Urotropin in Cystitis.—DR. C. POSNER considers that in connection with local treatment of cystitis the internal administration of urotropin is most important, and in certain especially chronic cases the drug should be used to the exclusion of instrumentation. Urotropin will be found also an effective preventive of catheter infection. Unfortunately, it seems to have little influence upon tuberculous or gonococcal infections of the bladder, but in inflammations due to bacillus coli or to staphylococci it is effective, and the reaction of the urine does not affect its action. The various drugs which have been exploited as substitutes for urotropin seem in no way superior, and certain of them may cause harmful effects.—*Berliner klinische Wochenschrift*, 1905, No. 2, p. 41.

[Clinically, in many instances of tuberculous cystitis, the bacillus of Koch cannot be found in the urine so long as urotropin is administered. —R. W. W.]

Extirpation of Adenoids in Exophthalmos and Chorea.—DR. B. HALZ, in two children, aged, respectively, seven and five years, affected with exophthalmos, without other signs of exophthalmic goitre, has observed the eye symptoms to disappear in ten days in the former after the removal of adenoids; in the latter a tonsillectomy was done without influencing the exophthalmos. Eight days later extirpation of the adenoids resulted in its disappearance in about five days. Two years later the first patient reappeared with recurrence of adenoids and of the

exophthalmos. Operation was followed by a second disappearance of this symptom. A third patient, a child, aged seven years, who had been suffering from chorea for about a month, was apparently cured by removal of the adenoids, which were of large size.—*Berliner klinische Wochenschrift*, 1905, No. 4, p. 91.

Epidural Cocaine Injections in Mucomembranous Enterocolitis.—Dr. J. Roux reports good results from this form of treatment in 2 obstinate cases; the first in a woman, aged twenty-seven years, who had been affected with the disease for two years; it was accompanied by severe pains, which previously only morphine had been able to abate. These were immediately diminished after the first epidural injection, and on the next day disappeared. The injections were continued once a week for the first month; every fifteen days for the second. Following the treatment all pain disappeared, the constipation became less obstinate, the feces no longer contained blood, and the general condition was much improved. The second patient was fifty-nine years of age, and an equally good result was obtained by similar treatment.—*La semaine médicale*, 1905, No. 9, p. 102.

Sodium Bromovalerianate.—Dr. Roue states that ordinary combinations of the bromides and valerian possess the disadvantage of a very disagreeable taste. This drawback may be avoided by a new organic combination of the two substances, a sodium bromovalerianate, which is entirely without toxicity in therapeutic dosage. It slows respiration, but does not affect its regularity or depth; it slows the heart, but does not change its rhythm, nor does it decrease blood pressure; it also diminishes reflex excitability and does not disturb either the digestive or the renal function. Therapeutically it should be preferable to potassium bromide in epilepsy, if for no other reason than that it may be given for long periods without producing unpleasant symptoms. The dosage is from 45 to 90 grains daily, either in solution or in capsules. The drug acts well in hysteria, palpitation, arrhythmia, the nervous symptoms of exophthalmic goitre, chorea, neurasthenia, and migraine. In pertussis it is superior to bromoform.—*Revue francaise de médecine et de chirurgie*, 1905, No. 9, p. 213.

Veronal.—Dr. Kress reports on the use of this drug in the obstinate insomnia of hysteria and neurasthenia. Its prompt action and the lack of unpleasant symptoms following its administration render it a valuable agent in these conditions. However, in 3 out of 12 neurasthenic patients, to whom 7½ grains of the drug were given nightly, after three or four days a cumulative action was noticed, which took the form of a pathological drowsiness, with defective assimilation and disinclination to leave the bed. Consequently, the author believes that we should always keep in mind the possibility of the appearance of such symptoms. Such disturbances indicate that the drug should be stopped at the first sign of cumulative action.—*Therapeutische Monatshefte*, 1905, No. 1, p. 37.

Antistreptococcus Serum.—Dr. F. Meyer has observed that the effects of antistreptococcic serum are not likely to appear until from twelve to twenty-four hours after the injection. For the first few hours the temperature remains almost constantly high; later it falls, and the patient

experiences an improvement in his symptoms. The author usually gives from 1¼ to 3½ drachms as a daily dose, and administers a short time before each injection a saline infusion of about half a pint. In severe anginas the treatment decreases the size of the tonsils; in diphtheria the serum is given in conjunction with antitoxin with good effect. Erysipelas is favorably affected by the serum, but the redness may increase following an injection. Scarlatinal sore throat is benefited, but the treatment does not seem to influence the course of the eruption. Excellent results are reported from the use of the serum in post-partum sepsis, but the author does not favor its administration in acute rheumatism. The serum causes no unpleasant effects, save a mild skin eruption.—*Berliner klinische Wochenschrift*, 1905, No. 8, p. 197.

The Administration of Tuberculin by Mouth.—Dr. Freymouth, in controversion of the statement of Koch, has proven that tuberculin is, under certain conditions, active when given by mouth and produces effects similar to those of injections of this agent. The minimum active dose by mouth is 0.075 to 0.150 minims given on an empty stomach and in keratin-coated capsules, with a little sodium bicarbonate to neutralize the gastric acidity. The reaction in some cases is as pronounced as that following an injection, and there seems to be present in the individual a relation between the sensitivity to tuberculin by mouth and the same substance given under the skin. The author considers it unfair, however, as yet to compare the therapeutic results of tuberculin given by these two methods, but believes that researches which he purposes to institute will show them to be identical.—*Münchener medizinische Wochenschrift*, 1905, No. 2, p. 63.

Gastric Intolerance in Young Children.—Dr. M. E. Terrien remarks the occurrence of marked gastric disturbances in children without intestinal manifestations. He distinguishes three causes: dyspepsia, acetonæmia, pyloric stenosis. The therapeutic indications are nearly the same in all. To arrest the emesis only water should be allowed at first—ice-water—a teaspoonful every half-hour, and hot applications to the epigastrium. Lavage may sometimes be necessary, and injections of artificial serum may be employed. When vomiting has become frequent, vegetable broths, salted, are allowed. When emesis has ceased, a bouillon, containing a coffeespoonful of farina to each three ounces, is given, to which, after a few days, a little milk may be added, and if well borne it may be gradually substituted for bouillon. The quantity of sugar in the milk must be carefully regulated. If the milk is not tolerated, we should return to the vegetable broth, and after a few days try buttermilk. If these means fail and pyloric stenosis is suspected, the case becomes one for the surgeon.—*Le bulletin médical*, 1905, No. 11, p. 119.

Gonorrhœa in the Female.—Dr. L. Archambault recommends in vulvovaginitis copious injections twice a day of 1:2000 to 1:4000 potassium permanganate, or of mercury bichloride 1:2000, followed by a dressing of 5 per cent. ichthyol or 25 per cent. thigenol in glycerin. Resorcin, in doses of 15 grains, may be given internally. Twice a week silver-nitrate solution should be used to swab the inflamed mucous membrane, and following this a powder of alum, 3 parts; tannin, 2 parts,

should be insufflated. Frequent bathing and other hygienic means should be employed. If there is complicating metritis, dressings of ichthyol, 10 parts; iodoform, 5 parts; glycerin, 200 parts, should be used. Local applications of tincture of iodine or of zinc chloride, 1: 50, may be employed, and intrauterine injections of about an ounce and a half of either of the following solutions: alumnol, 2½ parts; tincture of iodine and alcohol, each 25 parts; or dermatol and gum arabic, each 2 parts; water, 25 parts. Intrauterine bougies of dermatol, 8 grains; lanolin, 150 grains; white wax, 30 grains, are useful, as are tampons of beer yeast, which act by liberation of lactic acid. Urethritis should be treated by the balsams, the alkalines, and by irrigations of silver nitrate or protargol solutions, a 1 per cent. aqueous solution of thallin sulphate.—*Journal de médecine de Paris*, 1905, No. 9, p. 86.

The Finsen Light in Leprosy.—Dr. Lesser finds that in all cases of leprosy the Finsen light retards the course of the disease, but at present it is impossible to say that it will definitely cure it. The cosmetic results are better than with any other treatment, and, while influencing the lesions, the rays do not affect the surrounding tissues. In cases where the Finsen treatment seems insufficient, it should be combined with other therapeutic means—the *x*-rays, hot-air, caustics, etc.—*Revue de thérapeutique*, 1905, No. 4, p. 139.

The Treatment of Ocular Syphilis.—Dr. J. A. Abreu de Fialho has used intramuscular injections of mercury biniodide in a large number of cases of specific eye disease with most satisfactory results. In beginning the treatment one should provide against the possibility of an unusual susceptibility on the part of the patient to mercurials by giving small doses, and in order to make the injection as little painful as possible the author makes use of the following concentrated solution: mercury biniodide, 0.3 part; potassium iodide, 0.6 part; water, 19 parts. The beginning dosage for a man weighing 115 pounds should be about 2½ drachms of this solution, and may be increased as its action is noted.—*La semaine médicale*, 1905, No. 10, p. 115.

Zinc Peroxide in Varicose Ulcers.—Messrs. de Beurmann and Tanon report that the use of zinc peroxide (ZnO_2) in infected and sluggish wounds is attended with excellent results; after experimentation they have found the best means for exhibition of the drug to be a 20 per cent. ointment in white vaselin. The ulcer is cleansed with hydrogen peroxide, covered with the ointment, and dressed with sterile gauze. Such a dressing is allowed to remain for from three to six days, depending upon the degree of the infection. In all cases the authors have obtained a complete disappearance of suppuration and odor, and a rapid cicatrization.—*Revue française de médecine et de chirurgie*, 1905, No. 6, p. 137.

Balsam of Peru and Silver Nitrate in Leg Ulcers.—Dr. Pettreto has obtained excellent results in cases of sluggish leg ulcers by the use of the following ointment: silver nitrate, 1; balsam of Peru, 20; ointment, 300. Dressings of this will improve most cases, will lessen the suppuration, and cause a rapid growth of epithelium over the denuded areas.—*Le progrès médicale*, 1905, No. 8, p. 124.

Hot Air in Treatment of Eustachian Catarrh.—Drs. M. Lermoyez and G. Mahu state that the insufflation of hot air (160° to 195°) lessens the congestion and reduces the secretion of mucous membranes. The treatment is especially effective in rhinitis with nasal obstruction, in hypersecretion, and in acute coryza. The authors also recommend the treatment by means of hot air in otalgia secondary to nasopharyngeal affections and in stenosis of the Eustachian tube. Under the treatment the walls of the tube retract and the air pressure becomes normal. The necessary apparatus consists of a coil of tubing heated by a Bunsen burner through which air under pressure is passed. To this coil a hard-rubber Eustachian catheter, previously heated by passage through it of the hot air, is attached. Each treatment should last one to two minutes, during which short insufflations, lasting five to ten seconds, with intervals of two to three seconds, are given. Usually five to six treatments at intervals of one or two days are sufficient to relieve the patient. Spraying with 1 per cent. cocaine solution before the procedure lessens its unpleasantness.—*La presse médicale*, 1905, No. 2, p. 11.

The Treatment of Bronchopneumonia in Infants.—Dr. Theodor Zangger has treated 10 cases without mortality by sitz-baths at from 83° to 86°, gradually reduced to 75°. One or two baths daily were given; their duration was from three to six minutes, and during them friction was employed. The fever disappeared in from one to four days, and with it the other symptoms ameliorated. The water should not reach higher than the chest. After the bath the patient is wiped with a warm towel, and in the intervals of the procedure compresses wet with water at 60° are applied to the chest every one or two hours. Before and after the bath the patient is given milk, to which a little black tea is added in the later stages of the infection. Since bronchopneumonia is so prone to follow whooping-cough and measles, the author institutes the baths in these affections as a prophylactic measure, and with good results. The baths are said to increase blood pressure and expectoration and to have a laxative and diuretic action. —*Correspondenz-Blatt fur Schweizer Aertze*, 1905, No. 1, p. 7.

Adrenalin in Local Anæsthesia.—Dr. Hildebrand considers that the combination of cocaine and adrenalin produces a complete local anæsthesia. With the combination the duration of the anæsthesia is longer and the toxicity of the cocaine is diminished. For anæsthesia of mucous membranes, a few drops of 1:1000 adrenalin chloride solution are added to 5 per cent. cocaine solution. With this mixture the mucous membrane to be anæsthetized is tamponed, and in about five minutes a complete insensibility is obtained. To anæsthetize the bladder 0.1 to 0.2 per cent. saline-cocaine solution, to which 1.5 grains of suprarenin have been added, is used. The bladder is injected with this solution, which is allowed to remain for a quarter of an hour, and is then passed naturally. To anæsthetize the skin by hypodermic injection, 10 drops of adrenalin solution are added to 7½ drops of cocaine solution, and the mixture is injected in a circle about the inflamed area. The author does not advocate spinal anæsthesia, even though the addition to the solution used of adrenalin deprives the procedure of certain disadvantages.—*Berliner klinische Wochenschrift*, 1905, No. 1, p. 16.

Therapeutic Utilization of the Chemical Reaction in the Intestinal Tract.—Dr. J. Goldschmidt has administered to a patient suffering with tuberculous enteritis 15 grains of ichthoform three times a day. Two hours after each dose 1 drop of the tincture of iodine was given. The patient, whose temperature had previously been normal, after two days exhibited a temperature of 103.3°. The pyrexia disappeared when the medication was stopped. The author considers that a chemical combination takes place in the digestive tract and that this is intensified by the nascent state of the elements taking part in it, and that further research may result in an advance in the treatment of syphilis, the action of the iodine being intensified by its administration in the nascent state instead of in the form of an iodide.—*Therapeutische Monatshefte*, 1905, No. 1, p. 37.

Adrenalin in Gastrointestinal Hemorrhages.—M. Schlesinger has treated 2 patients by this agent. The first, a hæmophilic, suffered from grave intestinal hemorrhage, which was stopped by the hourly administration of 10 drops of a 1:1000 adrenalin chloride solution hourly. Under the influence of this agent the coagulability of the blood was increased, which was shown by the action of drops of blood drawn from the skin by a pin-prick. The second patient was suffering from Werlhoff's disease, with subcutaneous and submucous hemorrhages, as well as marked hæmatemeses. These last ceased under the administration of adrenalin given as in the preceding case. In other cases of gastric hemorrhage the author has found this agent valuable, but he considers it in no way superior to other methods of treatment in the control of hæmoptysis.—*Journal de médecine de Paris*, 1905, No. 1, p. 1.

The Salicylates in Inflammation of the Upper Respiratory Tract.—Dr. F. Halbhuber recommends the administration, two or four times a day, of a capsule containing 12 grains of sodium salicylate and 4 grains of antipyrin in acute rhinitis and pharyngobronchitis, with fever and headache. In subacute cases with chronic pharyngitis and irritation of the throat he advises the use of 22 grains of sodium salicylate a day, together with three or four capsules, each containing Dover's powder, 1½ grains; sodium bicarbonate, 1½ grains, and powdered acacia, 6 grains.—*Wiener medizinische Presse*, 1905, No. 1, p. 24.

Urotropin and General Paralysis.—Mr. N. F. MacHardy reports 2 instances of paresis, 1 associated with tabes dorsalis, which seem to have been cured by urotropin. The first case, a man aged forty-four years, had suffered from tabes for two years, and for one week from delusions of grandeur. He was observed from October 11, 1902, to October 1, 1904. His symptoms gradually increased in intensity until November, 1903, when he was put on urotropin, 5 grains per day. This was continued, the symptoms meanwhile gradually ameliorating until April 20, 1904, when the dosage was increased to 15 grains. Progress toward recovery continued until, on August 1st, he appeared only nervous. Two months later he showed no signs of recurrence. The second case, aged thirty-eight years, when first observed, had slurring speech, exaggerated knee-jerks and ankle clonus, twitching of facial muscles, and led a catheter life. Urotropin, 2½ grains twice a day,

caused an improvement, but later had to be stopped on account of hæmaturia. The symptoms began to reappear. On renewal of administration of urotropin improvement was rapid; less than two months from the first observation the patient was apparently well, and after a month more was discharged cured. About two months later was still well and continuing the urotropin. The author states, in conclusion, that the influence of the drug was very marked from day to day, and that signs of relapse disappeared upon increasing the dosage. These 2 were the only cases treated, and it seems that the method is deserving of a trial.—*British Medical Journal*, 1905, No. 2300, p. 185.

OBSTETRICS.

UNDER THE CHARGE OF

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Spontaneous Rupture of the Uterus.—OLSHAUSEN (*Zentralblatt für Gynäkologie*, 1905, No. 4) reports the case of a patient who, in her seventeenth labor, had spontaneous rupture of the uterus. The child was in vertex presentation, and the pelvis was ample in size.

Upon section there was blood in the abdominal cavity; a long rent was found in the posterior wall of the uterus extending to the vagina. His explanation of the uterine rupture was that the patient had a very well-marked prolapse of the anterior vaginal wall, which allowed the uterus to fall forward during labor, thus directing the presenting part against the uterine wall on its posterior aspect instead of into the brim of the pelvis.

Cæsarean Section for Dystocia following Myomectomy.—HUBBARD (*American Journal of Obstetrics*, March, 1905) reports the case of a patient who had myomectomy performed for two fibroids upon the anterior face of the uterus. After this the womb was fastened forward by two strands of kangaroo tendon, and during convalescence a mural abscess developed, which extended to the wounds on the anterior aspect of the uterus. The patient subsequently became pregnant in 1903 and was seen in labor, the membranes having ruptured two days previously. On examination the os was found to the left of the median line, the axis of the uterus lying almost transverse. Under ether it was impossible to cause the head to present or to apply forceps, and the child was extracted by version, an arm and leg being fractured. The child survived but a short time. A small fibroid was felt to the right of the fundus at this time.

Pregnancy again occurred in 1904, and the patient was admitted to the Boston Lying-in Hospital for section. Her pelvis was apparently normal, and the fœtus was again transverse, the breech being in the right iliac fossa and the head under the ribs on the left.

On section the placenta was beneath the uterine incision, and the child was delivered after incising the placenta. On the anterior surface of the uterus was a slight depression and scar, at the right cornu a small subserous fibroid. A month after operation a cicatrix was found on the left side of the vagina running to the cervix. There was a stellate tear in the cervix, but the neck of the womb lay in the middle line. The fundus was about one and a half inches to the right of the median line. Mother and child made a good recovery.

Although the title of the paper draws attention to dystocia following myomectomy, the real cause of the dystocia was the ventrosuspension. While dystocia after myomectomy has not been reported, dystocia after ventrofixation has frequently been seen and has required abdominal section for its relief.

The Growth of Lutein Cells during Pregnancy.—SEITZ (*Zentralblatt für Gynäkologie*, 1905, No. 9) has examined 36 ovaries removed from patients in various stages of pregnancy, from the second to the tenth month, and also during the puerperal period. He found in all of these hyperplasia of the tissue surrounding lutein cells in the follicles of the ovary. These follicles, he believes, show plainly the growth of lutein cells. He finds that the phenomenon is the same whether the pregnancy is a normal one or whether a vesicular mole is developed. He found these cells as early as the second and third months of pregnancy, and also in the middle and at the end of gestation. In a pregnancy of six weeks they were not present distinctly. In a tubal gestation of between six and seven weeks they had not completely developed. Regarding the function of these cells it is difficult to assert positively.

Rupture of the Uterus.—VALENTA (*Zentralblatt für Gynäkologie*, 1905, No. 9) reports 2 cases of rupture of the uterus, and has observed 14 cases of this accident.

His first case was a multipara who had a transverse position and shoulder presentation, and in whom the extraction of the child was attended with considerable difficulty, the physician attempting the operation being unable to extract the head. On admission to the hospital the patient's pulse was 120; there were no uterine contractions, and her temperature was normal. The head was easily extracted, but followed by severe bleeding. On examination the uterus was completely ruptured and the placenta was in the abdominal cavity. The point of rupture was hastily tamponed, and, as hemorrhage was not controlled by the tampon, abdominal section was performed. There was a rent 10 cm. long in the uterus, and the womb was amputated with extraperitoneal treatment of the stump. The patient rallied after the operation, and the stump was removed on the eleventh day. An exudate formed in the right parametrium, which disappeared after two weeks. The patient made a good recovery.

In the second case an attempt at version with transverse presentation was followed by bleeding, the attempt being unsuccessful. On admission to the hospital the uterus was evidently ruptured, and abdominal section was at once requested. This was declined by the patient. On vaginal examination the right arm of the fœtus and a mass of coagulated blood were found in the vagina. There was a rent in the posterior vaginal wall. The hand passed directly into the abdominal cavity and

came upon coils of intestine in the posterior portion of the uterus. Decapitation was performed when the intestine prolapsed. It was impossible to find the placenta, and a tampon was inserted to stop the hemorrhage. The patient then consented to abdominal section. The uterus was found torn across from the vagina, the tissues behind the cervix being widely torn. The stump was treated outside the peritoneum, and the peritoneum stitched above it. The rent in the posterior vaginal wall was closed from above. A packing of gauze was carried into the vagina against the point of rupture. The patient rallied from the operation, but her recovery was complicated by pelvic abscess, which ruptured and drained through the pelvis. She ultimately made a good recovery.

On examining the records of the clinic at Laibach, Valenta found that there had been 14 cases of complete rupture of the uterus during six years, in 1350 labors, or a percentage of 1.02. Three of these recovered without operation; 8 cases were operated upon by him; 3 recovered and 3 died without operation.

It is of the utmost importance that operation be performed as soon after rupture as possible. The writer quotes Klein's report of 149 cases of uncomplicated rupture of the uterus, with 56 per cent. recovery after operation. In 198 cases of uncomplicated rupture of the uterus treated, without operation, by a compression bandage, the use of a tampon, or drainage, the recovery rate was 48 per cent. When the bladder as well as the uterus was ruptured the mortality was 87 per cent. Treatment by drainage gave a mortality of 17 per cent.

SCHMID, in Schauta's clinic, reported and collected 179 cases of uterine rupture, with a mortality of 50 per cent. in cases operated upon for complete rupture. In those treated by drainage the mortality was 34.5 per cent. Walla quotes 40 per cent. recoveries after operation in the clinic at Budapest. ZWEIFEL, in 29 cases at the Leipzig clinic, had a mortality of 62.5 per cent. Some of these cases were admitted exsanguinated.

The total extirpation of the uterus has been recommended in these cases, but Valenta prefers suprapubic amputation, with the extraperitoneal treatment of the stump.

Cæsarean Section for Retrocervical Myoma.—MICHEL (*Zentralblatt fur Gynakologie*, 1905, No. 6) reports the case of a multipara, aged forty-one years, whose previous labors, with the exception of the last, had been normal. At the last labor a fœtus presented abnormally, but the midwife succeeded in delivering the patient. During the labor under consideration the uterine contractions were feeble and infrequent. When the patient was seen her pulse and temperature were normal; the fœtus was in vertex presentation, second position. The head was movable above the pelvis and the heart sounds were good. Upon internal examination the pelvic cavity was found largely occupied by a hard tumor the size of a fetal head. Anteriorly and above the symphysis the anterior lip of the cervix could be felt. The posterior lip could not be found. Two fingers could be carried through the cervix, when the membranes were found unruptured and the head above freely movable. An unsuccessful effort was made to push the tumor up. Upon abdominal section the uterus was emptied by transverse incision, and a vigorous child easily extracted. The tumor was found to be a myoma attached

to the posterior portion of the cervix. In view of the age of the patient and her prolonged labor, total extirpation of the uterus was performed. The patient made an uninterrupted recovery. The child was well developed and survived an acute pemphigus, which appeared on the second day after birth.

Appendicitis during Pregnancy.—HEATON (*British Medical Journal*, March 4, 1905) reports 4 cases of appendicitis complicating pregnancy, and has collected in addition 20 others in the literature of the subject.

Heaton's first case was that of a young primipara three months' pregnant, with acute appendicitis. There was a hard, tender, inflammatory mass in the right iliac fossa. Under rest in bed and medical treatment this subsided; the patient went to term and had a normal confinement. She was advised to have the appendix removed after her recovery from confinement, but did not consent. The second case was that of a woman in her second pregnancy, two months advanced, who had a violent attack of appendicitis. There was an extremely tender mass in the right iliac fossa. On vaginal examination this could be detected on the right side and above the uterus. On abdominal section a localized abscess was opened outside and below the cæcum, which was thoroughly drained. The patient made a good recovery, and the abscess cavity gradually closed. Pregnancy was uninterrupted, but at the time of writing had not yet reached full term.

The third case was that of a patient five months advanced in her second pregnancy, with severe appendicitis ushered in by a chill. Under rest in bed and opium the symptoms subsided. Five days afterward she had sudden pain in the lower half of the abdomen, with collapse. The cervix was dilated, and a five months' fœtus was readily delivered. General peritonitis followed, and on opening the abdomen the peritoneal cavity was found full of bloody fluid, the intestinal coils distended and glued to each other. The right ovary and tube were greatly swollen and injected and were removed. Behind the cæcum and colon there was a large abscess and in it a loose fecal concretion. In spite of free drainage, the patient died. The appendix upon autopsy was gangrenous and perforated at its extremity.

His fourth case was that of a multipara who, when seven months' pregnant, had a rigor and vomiting, with pain in the right iliac region. At the end of the week labor was induced and a dead fœtus delivered. After this the patient was severely ill for three months, while an abscess burst into the bowel and a large quantity of pus was discharged into the rectum. On admission to the hospital abdominal section was performed and the abscess was opened into the pelvis. It lay behind and to the right side of the uterus. The patient made an excellent recovery, and two years later a second abscess formed and was opened through the old scar. From this she again recovered. Later she came to the hospital for the third time with an abscess in the right iliac fossa. Upon opening this a fecal concretion was found in the pus. The patient finally made a complete recovery.

Of the 24 cases collected, 6 were non-suppurative. These recovered without operation, although the pregnancy was interrupted in several. In 18 suppuration occurred, and of these 9 recovered and 9 died, a mortality of 50 per cent. In only 4 of these was the pregnancy uninterrupted, giving a percentage of 78 in which abortion occurred after the

formation of pus. The necessity for immediate operation in these cases is self-evident.

Heaton also calls attention to appendicitis simulating ovarian disease. He reports 3 cases, in 1 of which the appendix was adherent to the ovary on the right side. In the second cystic ovarian disease was present with appendicitis, and in the third the appendix was adherent to the ovary and was entirely intrapelvic. These patients made a complete recovery after operation. Heaton believes that the surgeon should make it a rule in removing the appendix in women, where no suppuration is present, to examine the right uterine appendages before the abdomen is closed, and to loosen or remove diseased ovarian or tubal tissue.

Placenta Prævia, with Rupture of the Uterus at Seven Months.—Lea (*Journal of Obstetrics and Gynecology of the British Empire*, December, 1904) reports an interesting case of a multipara, seven months advanced in pregnancy, who went into a hospital after a sudden, painless, and profuse hemorrhage. On examination the cervix was high up, the os readily admitting one finger. The placenta lay in the lower uterine segment, partially detached. There was slight continuous hemorrhage, but no uterine contraction. A dilating bag was introduced without difficulty, and was removed four hours later, when the cervix admitted three fingers. The placenta was loose in the lower uterine segment, and the membranes unruptured. A macerated seven months fœtus was extracted without difficulty by version. The placenta was expelled and the uterus contracted firmly. The pulse, however, remained high and slight hemorrhage continued, and on examination a deep tear was found on the left side of the cervix. Hemorrhage from this tear ceased when sutures were applied. The uterus was firmly contracted; there was no external hemorrhage. About half an hour afterward the patient suddenly died.

On autopsy there was copious intra-abdominal bleeding through the linear rent in the posterior layer of the left broad ligament. A longitudinal tear had occurred on the left side of the lower uterine segment, between the layers of the broad ligament. This had formed a large hæmatoma, which had ruptured into the peritoneal cavity. The uterine muscle was pale, soft, and flabby. The rupture had occurred at the placental site. On looking into the patient's history there was evidence that she had been syphilitic. This had resulted in degeneration of the uterine muscle; while the implantation of the placenta into the lower uterine segment, the pressure of the back, and version and extraction had contributed this result.

Cæsarean Section at Term for Fibroid Tumor in a Double Uterus and Vagina.—Lyle (*Journal of Obstetrics and Gynecology of the British Empire*, December, 1904) reports the case of a patient who had had three abortions. Her fourth pregnancy went to full term. Delivery being delayed, examination revealed a fibroid in Douglas' *cul-de-sac*. The head was above the pelvic brim and could not enter. The child was delivered by section and the uterine wound closed with catgut stitches and the uterus lifted up, when the mass in Douglas' *cul-de-sac* was found to be a second myomatous uterus attached to the top of the vagina on its right side. There was an ovary and tube on its outer side, but none upon the inner. The peritoneum passed from the

posterior surface of the bladder between the two uteri and the anterior surface of the rectum. Suprapubic vaginal examination of the myomatous uterus was performed. On further examination the other uterus was found to have another tube and ovary placed upon its outer side. The patient made a good recovery.

On examination the vagina was found to be divided into two portions by a septum, and at the end of each canal was a distinct and separate cervix.

Three Cases of Cancer of the Cervix Complicating Labor and Advanced Pregnancy, the Patients Remaining Well Eleven, Eight and One-half, and Eight Years after High Amputation of the Cervix. —SPENCER (*Journal of Obstetrics and Gynecology of the British Empire*, December, 1904) reports the following cases:

Case I. Multipara, aged thirty-three years, was delivered by forceps, of a living child, at full term through a cancerous cervix a year before she came under Spencer's observation. The os dilated slowly, the posterior lip of the cervix dilating well, but the anterior lip, which was cancerous, did not yield. The membranes were ruptured artificially and labor progressed slowly. When the head was low in the pelvis a living child was delivered by forceps by very gentle traction. The placenta was expressed and the patient made a good recovery.

About a year after this the patient was admitted to the hospital in a pregnant condition, stating that she had had a discharge of blood and pus the last eight months. On examination the fundus reached three and one-half inches above the pubes. The anterior lip of the cervix was greatly enlarged by an ulcerating malignant growth. The posterior lip was free. The cervix was removed by high amputation, the vagina was incised in front of the cervix, the bladder pushed up and a similar incision made behind, and the base of the broad ligament was tied with thick catgut ligatures, and the part between the cervix and the ligatures divided with scissors. The tissue was freed higher up and the uterine cavity was opened anteriorly. The cervix was cut off and all bleeding stopped with a Paquelin cautery. The peritoneum was not opened. The vagina was packed with iodoform gauze and afterward boric acid douches and iodoform bougies were employed. She made a good recovery and left the hospital with her child in good condition. Subsequent pregnancy occurred, when the patient was delivered by Porro's operation, the ovaries and tubes being removed and the stump treated extraperitoneally. Eleven and a quarter years after the high amputation of the cervix the patient was in good health. The abdominal and vaginal scars were sound and free from hernia.

Case II. was that of a multipara, who, on admission to the hospital, complained of discharge and bleeding, with pain. The patient had had eight children, but no miscarriages. On admission the patient was in fairly good condition, the uterus at six and a half months, and the child was living. The cervix was high up and much enlarged, with a malignant growth on the posterior lip. The patient was advised to have labor induced and the cervix removed, and finally consented. She was then delivered by induced labor and artificial dilatation. The child survived about half an hour. The patient recovered from labor without serious complications, and between two and three weeks afterward the cervix was removed by high amputation, the mother making a good

recovery. More than eight and a half years after the removal of the cervix the patient was examined and found to be in good health. The scar was healthy, and there was no sign of recurrence.

Case III. was a multipara, who came to the hospital four and a half months after delivery, stating that she had had bleeding from the vagina daily since her confinement. On the left side of the posterior portion of the cervix there was a malignant growth. This was removed by high amputation, the patient making a good recovery. More than eight years after the operation the patient was well, the vaginal scars being normal and giving no evidence of malignant growth.

Spencer compares these results with those of the clinics at Leipzig and Berlin, giving the percentage of cures of 8.3 in operable cases, and in those too far advanced for radical cure treated by Cæsarean section, with a mortality of 80 per cent.

In his own experience he has had in hospital practice 6 cases of advanced pregnancy complicated with cancer of the cervix. Five of these were delivered through the vagina and operated upon during the puerperal period, and 1 of them was operated upon five months after parturition. All of these mothers recovered immediately from the operation. Two died seven months later, 1 twelve months later, and the other 3 remained well. One child died within half an hour, being premature; 1 died ten days afterward, while the remainder lived a number of months, sufficiently long to justify the method of treatment.

GYNECOLOGY.

UNDER THE CHARGE OF

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Radical Abdominal Operation for Cancer of the Uterus.—In a discussion of this subject before the Berlin Obstetrical and Gynecological Society, BUMM (*Zentralblatt für Gynäkologie*, 1905, No. 18) stated that he had had 75 cases, with 17 deaths, following operation; 56 patients had been under observation for over a year, of whom 23 were known to be well; 20 were free from recurrence at the end of two years. The speaker believed that a primary mortality of from 6 to 10 per cent. must imply an imperfect technique, since with the radical measures advocated by Mackenrodt a mortality of 20 per cent. was to be expected. With the extensive dissection necessary to remove all the diseased periuterine tissues it was impossible, he thought, to avoid a certain amount of septic infection, while there was always danger of injury to the ureters and bladder, not to speak of the risk of shock from such a prolonged operation. For these reasons he still gave the preference to the vaginal route in simple cases, in which the disease was confined to the uterus, and the organ was perfectly movable.

Bröse's mortality was stated to be 27 per cent. He favored abdominal section in all doubtful cases and when there was evidence of periuterine infiltration.

Mackenrodt, in closing the discussion, affirmed that the percentage of cures after abdominal extirpation of the cancerous uterus was over 30 per cent. It was necessary to select cases more carefully in order to obtain the best results. Preliminary curettement and applications of formalin gauze (twelve hours before operation) were the best safeguards against sepsis.

Early operation and the transverse abdominal incision were important factors in the success of the operation. The speaker preferred to resect diseased portions of the bladder rather than to dissect away the organ at the risk of having subsequent fistulæ. He no longer feared shock with his improved technique. According to his view, Olshausen's dictum with regard to the incurability of advanced cancer of the uterus applied to vaginal, but not to abdominal hysterectomy.

Ovarian Cysts of Unusual Size.—Zacharias (*Münchener med. Wochenschrift*, 1904, No. 31) reports the case of a woman, aged sixty-two years, from whom he removed 132 ounces of fluid by tapping an ovarian cyst. On account of the extensive adhesions and the patient's condition it was not deemed advisable to attempt its removal. The sac was drained, and she succumbed to exhaustion five weeks later.

The writer collected 14 similar cases of enormous cystomata of the ovary, in 7 of which ovariotomy was performed successfully; 5 with an immediate fatal result, while 2 patients succumbed after drainage of the sac.

Appendicitis in Women.—Rostowzew (*St. Petersburger Zentral Zeitung*, Nos 32 and 33) finds, with other observers, that it is often impossible to differentiate acute appendicitis from right adnexal disease, though the former has usually a more stormy onset. Pelvic abscess, torsion of cysts, extrauterine pregnancy, and thrombosis of the pampiniform plexus must all be eliminated. The writer even saw a case of dysmenorrhœa with apparent localized peritonitis, in which he was prevented from operating only by the rapid subsidence of the threatening symptoms. He advocates the removal of the appendix whenever the abdomen is opened for pelvic disease. He attributes the association of appendicular and adnexal inflammation rather to lymphatic infection through the medium of the ligamentum appendiculo-ovaricum than to direct contact.

Relation of Adenoid Tissue to Malignant Growths.—Meyer (*Sammlung klin. Vorträge*, No. 359) calls attention to the fact that this tissue is not only widely distributed throughout the body, but has an especial tendency to cell proliferation under the influence of peculiar disturbances common to the higher vertebrates. This is most marked in the case of the uterus. Cancer and sarcoma, according to his theory, are not due to specific organisms, but are of mycotic origin. The former are peripheral apolymphomata, the latter apolymphomata of the lymphatics. In the intestine and skin cancer is a similar apolymphomatous manifestation.

Result of Conservative Operations on the Adnexa.—BARONE (*Arch. di ost. e gin.; Zentralblatt für Gynäkologie*, 1905, No. 9) contends strongly in favor of the conservative treatment of all except malignant, tubercular, or purulent tubes and ovaries, since the mortality is so low and the curability was 87 per cent. in his hands. Menstruation persisted in 97 per cent., and pregnancy occurred in 12 per cent. The writer is also a firm believer in the efficacy of organotherapy in cases of amenorrhœa from chlorosis.

[The candid observer, while admitting the propriety of adopting conservative methods in the surgery of the adnexa, can hardly be as hopeful as the writer. Certainly, the combined experience of abdominal surgeons is opposed to the brilliant results reported by him.—H. C. C.]

OPHTHALMOLOGY.

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School Examination from an Ocular and Hygienic Standpoint.—GREEF (*Cent. f. prak. Augenh.*, February, 1905) reports the results of such examinations made in the higher schools of Berlin upon the initiative of the Minister of Public Instruction. Of 1437 pupils, follicles were found in the conjunctiva in 299, or 21 per cent. In one school the percentage was even as high as 31. According to the views of those who believe in the identity of follicular conjunctivitis and trachoma, these schools must be the seat of a flourishing epidemic of trachoma. Greef, however, is of the opinion that these follicles are not only to be sharply distinguished from trachoma and are capable clinically of being so distinguished, but that they do not even constitute a well-marked disease form. Such cases are found in schools everywhere and at all times and generally give rise to no complaints.

As regards the prevalence of myopia, all of the three higher schools examined showed about the same conditions, beginning with 15 per cent. in the lowest classes and reaching something less than 40 per cent. in the highest. Two of the schools presented the singular fact of a decrease in the number of myopes in going from the lowest to the next class. This inexplicable circumstance has been observed several times before.

Binocular Hemorrhages into the Vitreous.—FEJER, Bupapest (*Centralbl. f. Augenh.*, January, 1905), reports the case of a man, aged thirty-three years, who suffered rapid loss of sight from hemorrhage into the vitreous of both eyes, leaving only light projection. The sub-

ject was probably affected with lues, having been cured of an affection of the mouth eight years before by mercurial inunctions, although no present evidence of the disease could be found, unless a large number of lymphatic glands in the neck could be regarded as such. There was no evidence of disease elsewhere. The vitreous of both eyes was so full of opacities that no red reflex could be obtained. Hemorrhages re-occurred, which could be seen by focal illumination and even ordinary daylight. The treatment consisted of subconjunctival injections of a 2 per cent. solution of sodium chloride (painless under acoin), iodide of potash, and mercurial inunctions, and subsequently pilocarpine subcutaneously. No improvement resulted.

The author thinks there is no doubt that the etiological factor is to be sought in the diseased glands. But whether the hypertrophy was due to lues or scrofula could not be determined with certainty.

Hemorrhages into the vitreous are quite common, and are usually caused by traumatism. Of constitutional diseases, tuberculosis, pernicious anæmia, lues, and malaria are those which cause such changes in the vessels as to render their walls brittle. Disturbances of the general circulation also predispose to recurring hemorrhages of the vitreous. In such cases, Friedenwald and Fehr have demonstrated disease of the retinal veins; the former has observed perivasculitis with the ophthalmoscope; the latter has noted with the microscope thickening of the vessels, partial narrowing of the same through hyaline degeneration, and in places even entire obliteration. Some authors consider hereditary syphilis to be the cause of these changes.

Recurring hemorrhage of the retina and vitreous usually occurs, in youthful subjects between fifteen and twenty years of age. They are rare in older persons. The general organism frequently presents no pathological alterations elsewhere, the subjects being in apparently good health. The hemorrhage occurs first in the retina, later in the vitreous, is gradually absorbed, to be followed by a sudden recurrence. The prognosis is usually bad, leading to detachment and atrophy of the retina and vitreous.

Lymphoma Conjunctivitis (Parinaud's Infectious Conjunctivitis).—GOLDZIEHER, Budapest (*Centralbl. f. Aug.*, January, 1905), described a case of this disease in 1882 as lymphadenitis conjunctivæ; this was probably the first case of this affection reported; Parinaud's first 3 cases were not made public until 1889.

The characteristic mark of this disease is the development of large lymphomata in the cervical region, coincident with an extensive proliferation of granular and follicular structures in the conjunctiva of the same side, accompanied by a greater or less degree of inflammation, with chemosis and decided œdematous swelling of the lids. The affection, which appears to be very rare, may be easily mistaken for the granular form of trachoma, or perhaps tuberculosis of the conjunctiva. It is, however, an affection *sui generis*.

The disease is rapidly curable by surgical intervention (extirpation of the larger nodules with scissors, the smaller with galvanocautery). The expectant method (iodoform, astringents) is also frequently successful. Caustics are not to be employed. The internal administration of arsenic is followed by the rapid disappearance of the enlarged cervical glands; the latter rarely terminate in abscess.

Etiology. Parinaud assumed that the affection is caused by an animal contagium. This view does not seem to be established by subsequent observations; although a relatively large number of the individuals affected had to do with animals (butcher, drover). The affection, while manifestly infectious, is not at all contagious. As a rule, it is limited to one eye.

Bacteriological examinations have thus far failed to isolate the germ; but it is of importance to note that tubercle bacilli are not present. Microscopic examination seems to show that the process consists essentially of a chronic inflammation of the conjunctiva. It is found to be markedly different from trachoma.

Further and more extended examinations are required for a complete understanding of the disease in question.

Researches upon the Arterial Tension in Glaucoma.—FRAENKEL (*Arch. d'oph.*, January, 1905) publishes an exhaustive series of observations upon the subject, and comes to the conclusion that there is increase of arterial tension in subjects affected with glaucoma; other unknown pathogenic factors are also concerned. Attempt should be made to discover and render the latter precise.

Massage with Gray Oil as a Substitute for Subconjunctival Injections.—VACHER (*La clin. oph.*, 1904, No. 4) recommends massage with the above medicament, which is both unirritating and sterile. It has a double action: a mechanical one in regulating the intraocular circulation, and a medical, from the absorption of the mercury. Gray oil has the following composition: mercury, 27 per cent.; mercurial ointment, 6 per cent.; lanolin, 45 per cent.; olive oil, 22 per cent.

OTOLOGY.

UNDER THE CHARGE OF

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Labyrinth Necrosis. — GERBER (*Archiv für Ohrenheilkunde*, November, 1903) expresses the opinion in this paper, which is a general review of the literature of the subject, that the decrease in the number of reported cases of labyrinth necrosis during recent years is due to the improved methods of treatment of suppurative middle-ear disease, there remaining, of course, as sources of this complication such cases of panotitis as are incident to the inflammatory conditions accompanying measles, diphtheria, and scarlet fever.

The cases included in this compilation are, therefore, those in which the necrotic condition was the outcome of long-continued suppuration in the middle ear without recourse to remedial measures.

Nearly all of the cases of labyrinth necrosis recorded in the earlier years of otological observation were reported in the English literature by Wilde, Toynbee, and Shaw.

In 1875 Schwartze reported a series of 18 cases. In 1886 papers on this subject were published by Bezold; in 1894 by Bre, and in 1898 a compilation of 78 cases was made by von Oesch, while in the present communication Gerber not only reviews selected cases previously reported, but adds 1 of his own observation, making a total of 90 cases upon which he bases certain conclusions, the whole number of reported cases of labyrinth necrosis appearing to be comparatively small when the frequency of suppurative middle-ear disease is considered, Bezold, for instance, placing the occurrence of this complication as low as from 0.03 to 0.02 per cent.

There are, unfortunately, no sufficient statistics as to the etiological factors in labyrinth necrosis, but it is understandable that the acute exanthemata should play an important part in their causation, and in 27 collated cases the proportion was: scarlet fever, 15; measles, 3; tuberculosis, 3; pertussis, 1; while of the remaining 5 cases 3 were referred to trauma and 2 to coryza.

In the majority of cases the labyrinth infection is secondary to a chronic suppurative disease of the middle ear, but both Herzfeld and Gerber have, independently, reported cases of simultaneous infection of the labyrinth and middle ear other than true panotitis. As to the length of time between the primary and secondary infection the reports vary, mainly between three and twelve months, the longest interval reported being nineteen years.

Of all the reported cases included in this review the greatest number —37—occurred before ten years of age; 15 in the second decade; 16 in the third; 7 in the fourth; 8 in the fifth; 5 in the sixth, and 2 in the seventh, the acute exanthemata playing an important causative part in the first decade, Bezold being of the opinion, moreover, that the condition of the bone in childhood predisposes to sequestrum formation.

From the cases reported by Boeters and Bezold, especially, it would appear that males are more frequently affected than females, and that the left ear is more frequently the seat of labyrinth necrosis than the right. In a series of 71 cases the right ear was affected in 30 cases, the left ear in 37 cases, and in 4 cases the complication was bilateral.

Investigation as to the seat and extent of the necrotic invasion would tend to show the preponderating vulnerability of the cochlea. An analysis of 86 cases gave in 7, necrosis of the petrous portion of the temporal bone entire; in 17, necrosis of the entire labyrinth; in 26, partial necrosis of the cochlea; in 19, complete necrosis of the cochlea; in 10, necrosis of the cochlea and partially of the remainder of the labyrinth; in 5, labyrinth necrosis, the cochlea remaining intact, and in 2 cases the semicircular canals and the cochlea were distinctively affected.

In a series of 89 cases the cochlea was implicated in 79, while a general review showed that the cochlea exhibited the only points of necrosis in 53 per cent., and that the path of infection apparently lay through the foramen rotundum.

As regards the hearing power in the series of 90 cases, there was no record made in 25; loss of hearing was noted in 43 cases; partial loss

of hearing only in 22 cases, hearing by bone conduction remaining in some of them where it was absent by aërial conduction.

While it is generally inferred that destruction of the cochlea implies total loss of hearing, some of the unilateral cases were supposed to have a remnant of hearing power remaining, but in the 3 cases of bilateral labyrinth necrosis there was absolute loss of hearing to all sounds.

In a series of 65 cases in which facial paralysis accompanied the necrosis of the labyrinth, this symptom was complete in 50, permanent in 21, and transient in 13. These figures would make the ratio of facial paralysis about 77 per cent., but Bezold makes the proportion, from his observations, 83 per cent., and states further that when the necrosis of the labyrinth is considerable the paralysis is usually permanent; when the cochlea only is affected that this symptom is liable to be merely transient, and he also considers paralysis, following attacks of vertigo, a symptom of the beginning of sequestrum separation.

Disturbances of equilibrium are apt to occur with the onset of tinnitus, vomiting being an occasional accompaniment, but subjective noises are rare, because the cochlea is so frequently the part of the labyrinth first affected. Febrile symptoms and chills are rare, except in the event of systemic implication, and pain is comparatively infrequent, except during the discharge of a sequestrum, when it may be very severe.

Otoscopic examination usually demonstrated the existence of a chronic middle-ear suppuration with polypi, and often an extension of the destructive process outward, along the external auditory canal wall and upward into the antrum, mastoiditis occurring as a complication in 58.7 per cent. of all the cases.

Bezold considers deep-seated polypi as characteristic of the necrotic process, and cholesteatomatous formations as a fairly frequent accompaniment, though this is not the opinion of the author, and he is, furthermore, the authority for the statement that 20 per cent. of cases of labyrinth necrosis prove fatal. In 13 fatal cases the causes of death were: in 5, meningitis; in 2, meningitis and pyæmia; in 1, pyæmia; in 3, abscess of the cerebellum, and in 2, tuberculosis.

Aneurysm of the Carotid in the Middle Ear.—Dr. Schulte (*Monatsschrift für Ohrenheilkunde,* March, 1904) reports an interesting case of aneurysm of the carotid artery occurring in the middle ear.

The patient complained of a discharge from the ear which had persisted at intervals for the past six years. The examination showed a thickened membrane and retraction of the malleus. In the anterior quadrant was seen a perforation about the size of a pea, with a mass protruding resembling a polyp. Removal by a snare was tried, and was unsuccessful. Chromic acid was then used, and a dimpling under slight pressure was noticed. Incision was then done with a gush of arterial blood as a result. The hemorrhage was controlled by a tamponade for three days, when bleeding from both nose and ear began. The common carotid was ligated and the hemorrhage ceased for several days, only to recommence. Injections of a 10 per cent. solution of gelatin, forty grams at an injection, were given, and after this was repeated nineteen times the bleeding was fully controlled. The discharge ceased soon after the control of the hemorrhage.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

CHARLES HARRINGTON, M.D.,

ASSISTANT PROFESSOR OF HYGIENE, HARVARD MEDICAL SCHOOL.

An Epidemic of Typhoid Fever from Personal Contact.—Dr. Noetel (*Zeitschrift für Hygiene und Infectionskrankheiten*, June 24, 1904, Bd. xlvii.) describes an epidemic of typhoid fever which occurred in seven small communities within the district of Beuthen, near Russian Poland, and could be traced to none of the usual common causes. In a population of 65,000 there were 927 cases, with 84 deaths. Careful investigation exonerated the water supply, milk, and other foods, methods of sewage disposal, etc., from all blame. The population, chiefly Polish, had increased with unusual rapidity, owing to industrial development, and lived in an overcrowded condition; it consisted largely of coal-miners, who were industrious and well paid, but whose ideas about sanitation were of the most primitive sort. Noetel decided that the epidemic had been spread by direct contact from person to person, through carelessness and ignorance, fecal matter being not held in that aversion which obtains with people but slightly higher in the social scale. The epidemic was stopped by vigorous action on the part of the public authorities in the direction of disinfection of discharges, detection and isolation of ambulant cases, and general sanitation. Noetel emphasizes the importance of direct contact in the spread of typhoid fever, and also the fact that in such an epidemic it is necessary to determine as early as possible whether the cases are due to contact or to a common cause like polluted water. If the former, all the sick, even those only mildly so, should be hunted up and isolated for a long time in hospitals, and, in the mean time, energetic and systematic disinfection should be in force.

Bacterial Content of the Water of Public Swimming Baths.—From experiments carried out by Messrs. E. E. Glynn and J. C. Matthews (*Journal of State Medicine*, July, 1904, p. 400), it is seen that first-class baths contain fewer bacteria than second-class baths, and that salt-water baths contain fewer than fresh-water baths. True multiplication in salt water practically does not occur. The number of bacteria introduced into the water by the average first-class bather was found to be 4,000,000,000, and by the average second-class bather 6,000,000,000. For comparison, the number of bacteria given off in an ordinary slipper-bath was determined, and it was found to range from 2,000,000,000 to 25,000,000,000. The number of colon bacilli carried into the bath by the average first-class bather was about 2,500,000; the number introduced by the average second-class bather was exactly double. No attempt was made to classify the bacteria found, but on five plates containing 101 colonies, 23 of the latter proved to be staphylococci (staphylococci cereus albus 11, staphylococci pyogenes aureus 8, and staphylococci epidermidis albus 4).

Why Old Milk is Unwholesome, though Sweet.—According to PROFESSOR H. W. CONN (*Bulletin* 26, *Storrs Experiment Station*), old milk is never wholesome, even though it has been kept at a temperature of 50° F. and still remains sweet and uncurdled, since, while the bacteria which produce curdling do not develop abundantly at that temperature, many other species that produce undesirable changes grow day after day, and the milk may thus become far more unwholesome than sour milk. It is to the growth of these undesirable species that some of the cases of ice-cream poisoning are probably due, the cream being kept at a low temperature for several days until needed to supply a demand, and, meanwhile, becoming highly charged with the bacteria which elaborate poisonous substances.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Pernicious Anæmia: An Histological Study of Seventeen Cases.—GULLAND and GOODALL (*Journal of Pathology and Bacteriology*, 1905, vol. x. p. 124) conclude from their study that the essential feature of the disease, and the criterion in its diagnosis, lies in the fact that the condition is a megaloblastic anæmia. The widespread evidences of blood destruction occurring in liver, spleen, hæmolymph glands, and marrow, indicate abnormal vulnerability in the blood cells rather than a pathologically excessive hæmolytic action on the part of so many diverse tissues. The accumulation of iron in the liver is due partly to the disintegration of weakened or weakly blood corpuscles by endothelial cells and leukocytes, and partly (and to a much greater extent) to storage of iron, which is the product of red-blood corpuscles that have been disintegrated by phagocytes elsewhere. The accumulation of iron in the liver is not peculiar to pernicious anæmia, and is the normal result of the abnormal amount of blood destruction. There is no direct evidence of special disease of the intestine, and the intestine need not be the primary seat of toxin production, though in certain cases, and notably in bothriocephalatus anæmia, it probably is so. In some part of the body a toxin is produced which acts directly on the bone-marrow, interfering with normoblastic blood formation, leading to megaloblastic formation, and acting with a negative chemiotaxis upon leukocytes, especially of the neutrophilic variety. The large red-blood corpuscles produced by such a marrow, perhaps as much from their size as from inherent weakness, fall a ready prey to endothelial cells and leukocytes in the "hæmolytic" organs, notably hæmolymph glands, spleen, and marrow. It is

quite possible that certain individuals from congenital defect in the marrow may be specially prone to the disease, as there is little doubt that the megaloblastic degeneration represents a reversion to the fetal type.

The Value of Agglutination for the Diagnosis between Saprophytic and Pathogenic Streptococci.—FISCHER (*Centralblatt f. Bakteriologie u. Parasit.*, 1904, Band xxxvii. pp. 449, 597) concludes from the study of a large number of streptococci, both pathogenic and saprophytic, that from their agglutinative properties with anti-sera these two classes of organisms cannot be differentiated. A monivalent streptococcus serum produced by a streptococcus which has not been altered by passage through animals always agglutinates the homologous strain; but such a serum is not capable of agglutinating all streptococci. Heterologous, nearly allied strains, may be agglutinated by this serum in high and even higher dilutions than the homologous culture. As the relation between the strains becomes more distant, however, the immune serum shows gradual difference in agglutination. The varying action of a single streptococcus serum toward heterologous cultures offers further proof that there exists a multiplicity of varieties of the streptococcus.

The Destruction of Fraenkel's Pneumococcus in the Blood of Immunized and Hypervaccinated Animals.—TIZZONI and PANICHI (*Centralblatt f. Bakteriologie u. Parasitkunde*, 1905, Band xxxvi. p. 25) record some interesting experiments dealing with latent pneumococcic infections in immunized animals. They show that after intravenous injections of pneumococcus virus in immunized animals pneumococci may remain in the circulating blood for days, weeks, and even months (in one case 205 days), and during this time may be cultivated from the blood of the ear veins. It makes no difference whether the animals are actively immunized or undergo passive immunity by injections of antipneumococcic immune serum at the time when the virus is inoculated—the result is the same. Neither does the grade of immunity (complete or incomplete) seem to affect the result. The disturbances which are observed with incomplete immunity (fever, nervous symptoms, and wasting) are not associated with the sudden liberation of toxins by a rapid destruction of bacteria, but are the result rather of an incomplete neutralization of primarily and secondarily produced toxins. In partially protected animals a slight injury to some part of the body frequently determines the seat of a localized infection by pneumococci. The total and complete destruction of the bacteria in the blood depends more upon the quantity of virus injected than upon the grade of immunity produced. In the blood of such animals as the sheep and ass, animals not very susceptible to the pneumococcus, the destruction of bacteria is much more rapid than in the blood of rabbits, very susceptible animals. Finally, the pneumococci which are recovered from the blood of these animals present certain modifications in their morphology, cultural characteristics, and pathogenic properties, for their virulence is greatly attenuated. By a few transplants upon culture media, the characteristic growths and morphology return, while the passage of a culture through one or two animals renders it pathogenic.

The authors believe that these experiments may throw some light upon the origin of many of the complications of such acute infections as pneumonia and typhoid fever, and may go to explain recurrent infections such as are observed in rheumatic fever, etc.

The protection conferred by injections of pneumococci is looked upon as a true antitoxic immunity, and the explanation for the persistence, without apparent ill effect, of pneumococci in the blood of immunized animals is to be sought in the fact that the toxins produced by the circulating bacteria are neutralized, and at the same time the virulence of the bacteria is lessened and finally lost.

Third Report of the Caroline Brewer Croft Cancer Commission of the Harvard Medical School.—(*Journal of Medical Research*, 1905, vol. xiii., No. 2.) The first paper is by F. B. MALLORY, entitled, "A Contribution to the Classification of Tumors" (p. 113). It is the beginning of an attempt at the classification of tumors based on a more careful study of histological differentiation of cells and intercellular substances. This paper deals especially with the differentiation of tumors arising from the neuroglia, from smooth muscle, and from connective tissue; and the classification depends largely upon the characteristic fibrils produced by these tissues, both normally and in tumor cells. These fibrils are called neuroglia, myoglia, and fibroglia, according to the tissue from which they arise.

Special staining methods are used to bring out the structure of the individual fibrils and their histological differences are explained and pictured. All the fibrils touch or form part of the periphery of the cell protoplasm, but continue away from the cell in two directions, neither beginning nor ending in the cell which produced them.

The myoglia fibrils seem to run along other muscle cells or to join the fibrils formed by them, while the fibroglia and neuroglia fibrils seem to be free from the protoplasm of other cells.

Tumors arising from neuroglia, smooth muscle, or connective tissue cells are composed of cells that tend to differentiate in the same way as the corresponding normal cells do. This may be made out even in rapidly growing tumors, where the differentiation is least well marked when the tissue is properly fixed directly after removal at operation. The author considers that all tumors the cells of which tend to differentiate alike in cell and intercellular substance should be included under one heading and treated as a definite entity in order that common characteristics may be rendered prominent.

The second paper of the series is "On the Nature of the Cell Inclusions of Cancer," by ROBERT B. GREENOUGH (p. 137). The author has studied cell inclusions in 177 cases, using tissue from carcinomatous growths and from non-malignant lesions, including 23 cases of chronic cystic mastitis. The method employed was as a routine, fixation in Zenker's fluid and staining by iron hæmatoxylin (Mallory), followed by orange G and fuchsin. His own cases and those of Nösska, Nichols, and Honda, writers holding views upon this subject similar to the author, are tabulated. In the 481 tissues from both malignant and non-malignant lesions cell inclusions or Plimmer's bodies were found 217 times. In the 23 cases of chronic cystic mastitis which the author studied, cell inclusions were found in 16. They were found almost constantly in cancer of glandular origin, but were almost invariably absent in sarcoma, and do not occur in epithelioma. They were found in hypernephroma, non-cancerous lesions of the mammary gland, and occasionally in other gland epithelium in non-cancerous conditions. Various characteristics of the cell inclusions are discussed, and the author con-

cludes that their size, structure, and staining reactions are such as to justify the assumption that they are vacuoles in the cell protoplasm containing a material which is coagulated and shrunken by the use of tissue fixatives. He considers that the vacuoles are chiefly a phenomenon of cell secretion, but are also produced by phagocytosis and by degenerations of the nucleus. Secretion vacuoles found in certain noncancerous diseases of the mammary gland occupy a position between the nucleus and the lumen of the gland. Inclusions in adenocarcinoma occupy a similar position, while in more advanced carcinoma the gland lumen is lost, so the secretion vacuoles cannot escape. They remain, therefore, within the cancer cell to undergo further increase and ultimate degeneration.

Cell inclusions are more numerous in slowly growing cancer, because, as the author thinks, the function of cell secretion is lost in progressive anaplasia of cancer cells. He finds no reason to interpret the appearances found as of parasitic origin.

A "Study of the Effects of the Roentgen Ray upon Cancer," by VOSE and HOWE (p. 167), constitutes the third paper. They studied 120 cases treated by the *x*-ray and endeavored to use conditions as similar as possible with all. Besides the action of the *x*-ray upon cancer, its effects upon the normal skin, ulcerated surfaces, and scar tissue, and its analgesic effect are discussed.

They report in detail 14 cases of various kinds. These cases include 2 of diseases of lymph nodes, 1 of tuberculosis, and 1 of Hodgkin's disease, where there was great reduction in the size of the lymph glands due, the authors believe, entirely to the counterirritation produced by the *x*-ray. The reduction of the glands was not permanent in the Hodgkin's disease case. The other 12 cases include instances of both cutaneous and deep-seated cancer. From their histological study, the authors consider that cutaneous cancer treated by the *x*-ray undergoes a degeneration not peculiar to this form of treatment or distinguishable histologically from degeneration due to other causes. The vascular changes are limited to an endarteritis, but new formation of blood-vessels occurs if healing takes place. There is an increase in elastic tissue following exposure to the *x*-ray, and after its use mitotic forms are less abundant in the tissue cells.

The authors consider this treatment limited in its value to superficial growths, for destruction and exfoliation are the only means of cure. They consider that the *x*-ray has no selective action.

"Implantation of Tissue and its Relation to Cancer," by EDWARD H. NICHOLS (p. 187), forms the last paper of the report. The author records the results of a series of implantations of tissues in 62 animals. Both adult and fetal tissues were implanted, and, although some mesenchymal tissues were made use of, the experiments were conducted chiefly with epithelium. The work was undertaken to discover whether these tissues, set free from their normal environment, would take on an unlimited growth and form metastases. Although in some cases the implanted tissues proliferated to form tumor masses, in none was there unlimited metastasis. The author discusses quite thoroughly the general arrangements for the parasitic origin of cancer and finds in none of them anything conclusive in support of this hypothesis. The blastomycetic and the protozoön theory of the causation of cancer are reviewed in the light of the experiments of the author and others, and he finds very little experimental evidence for their acceptance.

He considers that there are a considerable number of clinical and experimental facts in support of the biological theories of the origin of malignant and benign tumors, as enunciated by Thiersch, Cohnheim, and Ribbert.

From his own experiments, Nichols concludes that when certain types of epithelium (epidermis), both adult and fetal, are removed from their normal position and implanted into another part of the same animal, they can maintain their "potentiality of growth," to retain their own peculiar character, and produce nodules analogous to dermoid cysts or more complicated teratomata. Highly differentiated epithelium never retained its power of growth. The "potentiality of growth" was found to be greater in fetal than in adult tissues, but in no case was infiltration of surrounding tissues or epithelial metastases seen. Certain fetal connective tissues (cartilage) were transplanted and were found to retain their potentiality of growth. The transplanted fetal tissues were found to have a tendency to reproduce the ultimate stage of their development, and not that stage at which they were transplanted.

The Agglutination of Paratyphoid Bacilli and Closely Allied Bacteria by the Serum of Typhoid Patients.—GRÜNBERG and ROLLY (*Münch. med. Wochenschrift*, 1905, No. 3, p. 105) have tested the agglutinating action of the serum from 40 cases of typhoid fever upon bacillus paratyphi types "a" and "b," bacillus coli, bacillus enteriditis (Gärtner), and bacillus botulinus. The diagnosis of typhoid fever was assured in 32 instances (80 per cent.) by a growth of typhoid bacilli from the blood during the attack of fever, while in the remaining cases, the typical course of the disease left no doubt as to the diagnosis. In 70 per cent. of the cases paratyphoid bacilli, as well as typhoid bacilli, were agglutinated, and in 35 per cent. of the cases the reaction occurred in higher dilutions with paratyphoid bacilli than with the typhoid bacillus. The possibility of a mixed infection by the two types of bacteria can be practically excluded, and the authors agree with Jürgens in considering the phenomenon as a group agglutination.

In 12 out of 22 cases (55 per cent.) bacillus coli was agglutinated, usually in low dilutions, but since Pfaundler has already shown that bacillus coli may react with the serum of normal individuals, the authors do not consider that their results with this organism are of great significance.

Agglutination was quite constant with bacillus enteriditis, but took place only three times with bacillus botulinus. As a result of these investigations the authors conclude that the Gruber-Widal reaction is not a highly specific phenomenon, since, under certain conditions, the serum of typhoid fever patients may agglutinate bacilli nearly related to bacillus typhosus, even in higher dilutions than the typhoid bacillus itself.

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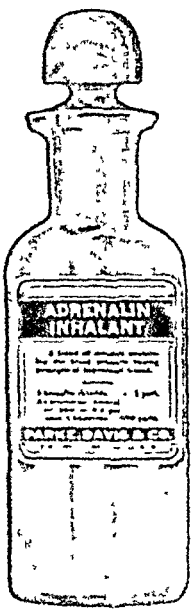
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(Laxative-Analgesic-Antipyretic)

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(Tonic-Laxative—Analgesic—Antipyretic)

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The Antikamnia Chemical Company
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Hay Fever

IN the opinion of many prominent rhinologists, laryngologists and general practitioners Solution Adrenalin Chloride and Adrenalin Inhalant meet the indications in Hay Fever better than any other agent.

SOLUTION ADRENALIN CHLORIDE

is a remarkably powerful astringent. In the treatment of Hay Fever it is usually diluted with four to five times its volume of physiologic salt solution. Sprayed into the nostrils by means of a hand atomizer, it promptly reduces the hyperemia and turgescence of the mucous membranes of the eyes, nose, etc., and checks the profuse secretions. It allays the nasal irritation and controls paroxysms of sneezing. Two or three applications a day ordinarily serve to keep the patient in a state of comparative comfort.

Supplied in ounce glass-stoppered vials.

ADRENALIN INHALANT

a more recent acquisition to our list of therapeutic agents, has been used with marked success in the treatment of Hay Fever during the past two seasons. It contains one part of Adrenalin Chloride to 1000 parts of a neutral oil. The best results are obtained by spraying it into the nasal passages from an instrument adapted to heavy, oily solutions. Requiring no dilution, it offers an especially convenient method of application, and its oleaginous base imparts an emollient effect that is particularly gratifying to the patient.

Supplied in ounce glass-stoppered vials.

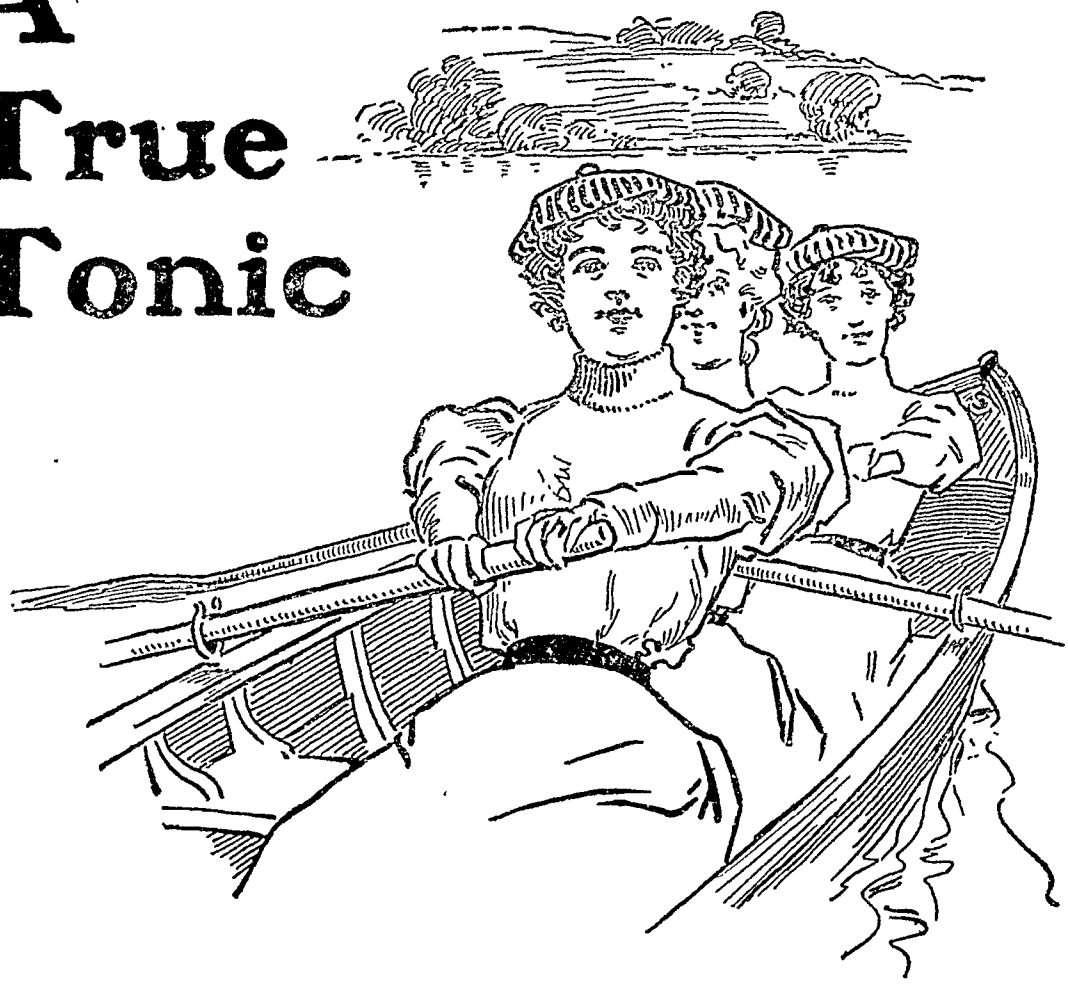
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